THE RELATIONSHIP BETWEEN PHYSICAL ACTIVITY AND THE RISK OF TYPE 2 DIABETES MELLITUS IN ELLISRAS RURAL YOUNG ADULTS AGED 22 TO 30 YEARS: ELLISRAS LONGITUDINAL STUDY

by

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DISSERTATION

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"*Education is what remains after what one has forgotten what one has learnt in school*." – Albert Einstein

"*An investment in knowledge pays the best interest."* – Benjamin Franklin

DEDICATION

I dedicate this dissertation to my late father (Pholala Alpheus Matshipi) who lost the battle with diabetes, for always encouraging me to never stop studying. I would like to express special gratitude to my mother (Mahlatsie Elizabeth Matshipi) and siblings (Emmah, Margreth and Matome Matshipi), who continuously encouraged and supported me through my studies. Furthermore, thank you to my dearest friend (Motlatsi Evans Lebea) for encouraging me to go for it. I would also like to thank the Ellisras Longitudinal Study team for offering moral support whenever I needed it.

DECLARATION

I declare that THE RELATIONSHIP BETWEEN PHYSICAL ACTIVITY AND THE RISK OF TYPE 2 DIABETES MELLITUS IN ELLISRAS RURAL YOUNG ADULTS AGED 22 TO 30 YEARS: ELLISRAS LONGITUDINAL STUDY is my own work and that all sources that I have used or quoted have been indicated by means of complete references and that this work has not been submitted before for any other degree at any other institution.

…….. ……

Full names Date

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ABSTRACT

Background

Type 2 diabetes mellitus (T2DM) is an increasing challenge globally, and is estimated to affect 439 million adults by 2030. This estimate is linked to an unhealthy lifestyle with characteristics such as low physical activity (PA) and high plasma glucose levels (PGLs). Studies associating PA with insulin resistance and diabetes among adults and adolescents have been conducted widely in developed countries. Such studies are scanty among rural populations, especially in Africa. Assessment of the burden of diabetes and associated lifestyle risk factors in developing countries is essential in order to encourage appropriate intervention strategies to counter the increasing prevalence.

Aim and objectives

The aim of this study was to investigate the relationship between PA and T2DM among rural young adults aged 22 to 30 years in Ellisras area in Limpopo Province, South Africa

Methods

A total of 713 young adults (349 males and 364 females) who have been part of the Ellisras Longitudinal Study participated in the current study. Physical activity data was collected using a validated questionnaire. After an overnight fast, participants provided fasting venous blood samples for determination of plasma glucose and insulin. Insulin resistance was estimated using the homeostasis model assessment of insulin resistance. Anthropometric measurements (waist circumference and height) were performed using standard procedures. Linear and logistic regressions were used to assess the relationship between PA, pre-diabetes, insulin resistance and T2DM; and the odds of having T2DM with low PA levels.

Results

The prevalence of physical inactivity was 67.3 and 71.0% for males and females, respectively. That of pre-diabetes was between 45.7% and 50.2%. The prevalence of diabetes was 9.6% for males and 10.1% for females while for insulin resistance was 22.9% for males and 29.3% for females. Linear regression found a significant

relationship ($p<0.05$) between physical activity and blood glucose (ß =5.715; 95% CI 4.545; 6.885), waist circumference (ß = 37.572; 95% CI 25.970; 49.174) and waist-to-height ratio (ß = 0.192; 95% CI 0.087; 0.296). Logistic regression found a significant ($p<0.05$) relationship between low physical activity and T2DM (Odds ratio = 2.890; 95% CI 1.715; 4.870) and insulin resistance (Odds ratio = 1.819; 95% CI 1.266; 2.614).

Conclusion

Physical activity is low in this population, and is independently associated with T2DM and insulin resistance.

KEY WORDS

Type 2 diabetes mellitus; pre-diabetes; insulin resistance; physical activity; young adults; rural South African population.

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LIST OF ABBREVIATIONS

ADA	American Diabetes Association
BMI	Body Mass Index
CVDs	Cardiovascular Diseases
DoH	Department of Health
ELS	Ellisras Longitudinal Study
FPG	Fasting Plasma Glucose
HOMA-IR	Homeostatic Model Assessment for Insulin Resistance
IDF	International Diabetes Federation
IFG	Impaired Fasting Glucose
IGT	Impaired Glucose Tolerance
IPAQ	International Physical Activity Questionnaire
IR	Insulin Resistance
ISAK	International Society for the Advancement of Kinanthropometry
MET	Metabolic Equivalents
MODY	Maturity-onset Diabetes of the Young
OGTT	Oral Glucose Tolerance Test
PA	Physical Activity
SES	Socio-economic Status
SPSS	Statistical Package for Social Sciences
T2DM	Type 2 Diabetes Mellitus
TEM	Technical Error of Measurement
WAT	White Adipose Tissue
WC	Waist Circumference

WHF	World Heart Foundation
WHO	World Health Organisation
WHtR	Waist to Height Ratio

CHAPTER 1

PROBLEMS AND AIMS OF THE STUDY

1.1. Problem statement

1.2. Rationale

1.3. Aim and objectives of the study

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1.1. PROBLEM STATEMENT

The prevalence of diabetes is on the increase across the globe, and is considered to be one of the leading causes of death world-wide (Guariguata et al., 2014). Grace et al. (2007) asserts that type 2 diabetes mellitus (T2DM) is caused by hereditary as well as lifestyle characteristics such as a poor diet and a sedentary behaviour. Unhealthy lifestyle habits such as over-eating, high carbohydrate diets, and low physical activity (PA) levels may possibly lead to an increase in blood glucose levels over time, thus increasing the likelihood of developing T2DM in adulthood (Guariguata *et al*., 2014). In addition, poor weight management, such as obesity, has been linked to T2DM (Rana et al., 2007). Effects of a modernised lifestyle are contributing to the increase in T2DM (Monyeki *et al*., 2012). The need to investigate the existence or risk of T2DM development in Ellisras young adults is deemed necessary for either prevention or treatment. A rural population, where there is a shift from an active lifestyle to a modern sedentary one (Popkin, 1999) was chosen in the Ellisras region, given the lack of information on T2DM in this population (Frankhuisen et al., 2001).

1.2. RATIONALE

Nair (2007) defines diabetes mellitus (DM) as a medical condition whereby the human body fails to properly metabolise glucose for use as energy, as a result of insulin resistance (type 2 diabetes) or insulin insufficiency (type 1 diabetes). The number of people with T2DM is increasing world-wide and is expected to be 592 million by the year 2035 (Bartoli et al. 2011; Guariguata et al., 2014). Hossain et al. (2007) adds that this increase is closely linked to the upsurge in obesity. Furthermore, Lloyd-Jones et al. (2009) noted that T2DM is a risk factor for vascular disease with 65% of all diabetic deaths being due to cardiovascular disease. Knowledge of the risk factors associated with T2DM is essential to treat and prevent future complications of the disease (Grace et al., 2007).

Physical activity is defined as any bodily movement that results in energy expenditure (Kemper, 2004). Physical activity plays an important role in the control of both weight and plasma glucose, thus reducing the likelihood of developing T2DM (Sanz et al., 2010). Physical activity also improves insulin sensitivity, which helps with the uptake of glucose by cells (Sanz et al., 2010). Buman et al. (2014) found that reallocating 30

minutes per day of sedentary time to light-intensity PA was associated with 2.4% reduction in insulin levels. Physical activity promotes the treatment of insulin resistance through a reduction in white adipose tissue (WAT) mass (Ross and Bradshaw, 2009). Accumulation of WAT results in the reduction of phosphorylation of insulin receptors in adipocytes, thereby, decreasing the expression of insulin receptor, subsequently leading to lower glucose uptake into adipocytes (Algenstaedt et al., 2004; Buren et al., 2003). Physical activity also lowers levels of glycated haemoglobin, which is a marker for T2DM (Dunstan et al., 2002). Little is known about the benefits of PA on the prevention and treatment of T2DM in the rural South African population.

Preliminary results of Ellisras Longitudinal Study (ELS) showed a decrease in PA over time in both girls and boys (Monyeki et al., 2007). It was also shown that T2DM did not exist in the sample population (Frankhuisen et al., 2001). However, the prevalence of obesity, overweight and hypertension was reported to be on the rise among the Ellisras children over time, indicating the possibility of the emergence of diabetes (Monyeki et al., 2008). There is a need to assess the burden of diabetes and its associated lifestyle risk factors in rural populations in order to encourage appropriate intervention strategies to counter the increasing prevalence.

1.3. AIM AND OBJECTIVES OF THE STUDY

The aim of this study was to investigate the relationship between PA and T2DM among rural young adults aged 22 to 30 years in Ellisras area in Limpopo Province, South Africa.

The objectives of the study were to:

i. investigate the prevalence of physical inactivity among ELS young adults aged 22 to 30 years.
ii. investigate the prevalence insulin resistance among ELS young adults aged 22 to 30 years.
iii. investigate the prevalence T2DM among ELS young adults aged 22 to 30 years.

iv. assess the prevalence of overweight and obesity using a variety of anthropometric indeses among ELS young adults aged 22 to 30 years.

v. assess the relationship between PA and levels of glucose among ELS young adults aged 22 to 30 years.

vi. assess the relationship between PA and insulin resistance, obesity indices and T2DM among ELS young adults aged 22 to 30 years.

vii. determine the risk of having T2DM with low levels of PA.

1.4. SCIENTIFIC CONTRIBUTION

This study adds to the limited understanding of the relationship between physical activity and type 2 diabetes mellitus in rural African populations. The findings open a window for future studies to focus on intervention strategies aimed to reduce the risks of type 2 diabetes in rural South African populations. This study also aims to encourage improvement of lifestyle characteristics such as physical activity and weight management in the Ellisras community through feedback and recommending lifestyle adjustments for the prevention of T2DM. It complements the mission and vision of the World Heart Foundation (WHF) to reduce mortality from cardiovascular disease (CVD) by 25% in 2025.

1.5. STRUCTURE OF THE DISSERTATION

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CHAPTER 2

LITERATURE REVIEW

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2.1. INTRODUCTION

Diabetes mellitus is one of the most common chronic diseases in nearly all countries, and continues to increase in numbers and significance (Wu et al., 2014). The global prevalence of diabetes has nearly doubled since 1980, rising from 4.7% to 8.5% in the adult population (WHO, 2016). Over the past decade, diabetes prevalence has risen faster in low- and middle-income countries than in high-income countries (WHO, 2016).

The number of people with T2DM is projected to be 592 million by the year 2035 (Guariguata et al., 2014), and was already at 422 million in 2016 (WHO, 2016). It is suggested that this reflects an increase in associated risk factors such as being overweight or obese (WHO, 2016; Whiting et al., 2011) supports the suggestion by adding that economic development and urbanisation lead to changing lifestyles characterised by reduced physical activity (PA), and thus increased obesity. Diabetes is a growing concern as it can lead to complications such as heart attack, stroke, kidney failure, leg amputation, vision loss, nerve damage and even increase the chance of premature death (WHO, 2016).

As the prevalence of diabetes continues to increase and prevention of T2DM becomes a worldwide concern, there is convincing evidence that T2DM can be prevented or delayed through lifestyle modifications which include change in diet, increased physical activity and maintaining normal body weight (Knowler et al., 2002; Gillies et al., 2007).

Physical activity, such as household, occupational, and travel related activities, plays an important role in the control of both weight and plasma glucose, thus reducing the likelihood of developing T2DM (Sanz et al., 2010). This is made possible by the ability of PA to improve insulin sensitivity, which helps with the uptake of glucose by cells (Sanz et al., 2010). Although the improvement of the population's knowledge and awareness of the risk factors associated with T2DM is essential to prevent future complications of the disease (Grace et al., 2007), such information appears to be

scanty in rural populations where modernisation and industrialisation take place (Popkin, 1999).

2.2. CLASSIFICATION OF DIABETES

The American Diabetes Association (ADA, 2014) classifies diabetes into four categories, namely: Type 1 diabetes mellitus (insulin-dependent diabetes mellitus), T2DM (non-insulin-dependent diabetes mellitus), gestational diabetes (diabetes diagnosed in the second or third trimester of pregnancy) and specific types of diabetes due to other causes, such as monogenic diabetes syndromes (such as neonatal diabetes and maturity-onset diabetes of the young (MODY)), diseases of the exocrine pancreas (such as cystic fibrosis), and drug- or chemical-induced diabetes (such as in the treatment of HIV/AIDS or after organ transplantation).

Type 1 Insulin-dependent diabetes mellitus (which accounts for only 5 – 10% of people with diabetes), results from a cellular-mediated autoimmune destruction of the β-cells of the pancreas (Yoon and Jun, 2005). Markers of the immune destruction of the β-cell include islet cell autoantibodies, and autoantibodies to insulin (Couri et al., 2006). Also, the disease is associated with increased or decreased frequency of certain histocompatibility antigens on chromosome six and with islet cell antibodies (ADA, 2004).

Type 2 Noninsulin-dependent diabetes mellitus (which accounts for about 90 – 95% of people with diabetes), encompasses individuals who have insulin resistance, a condition in which cells fail to use insulin properly, sometimes combined with an absolute insulin deficiency (DeFronzo, 2009). There are no known aetiologies for this type of diabetes, although it is frequently associated with obesity (ADA, 2004).

Diabetes may be associated with certian conditions and syndromes/the type of diabetes caused by other conditions or found in increased frequency with other conditions such as diseases of the exocrine pancreas, genetic defects in insulin action and MODY (ADA, 2004). MODY is Type 2 diabetes that develops usually in type 1

age group. Four genetic defects have been associated with the condition, one of which is a mutation on the glucokinase gene (Park et al., 2015). Glucokinase catalyses the conversion of glucose to glucose-6-phosphate and eventually the metabolites that stimulate the insulin secretion in the β-cell (Froguel et al., 1992). Thus glucokinase serves as the glucose sensor for the β-cell. The defect means that increased levels of glucose are required to elicit the usual secretion of insulin (Park et al., 2015).

Gestational diabetes refers to the type of diabetes that develops or is diagnosed during pregnancy (ADA, 1979). Gestational diabetes is also defined as any degree of glucose intolerance with onset or first recognition during pregnancy (ADA, 2004). During pregnancy, the pancreatic function might not be sufficient to overcome the insulin resistance created by the anti-insulin hormones secreted by the placenta (oestrogen, prolactin, human placental lactogen, cortisol and progesterone) (Wilcox, 2005).

2.3. DIAGNOSIS OF TYPE 2 DIABETES MELLITUS

Diabetes may be diagnosed based on glycohaemoglobin test (A1C) criteria or plasma glucose criteria, either the fasting plasma glucose (FPG) or the 2-h plasma glucose (2-h PG) value after a 75-g oral glucose tolerance test (OGTT) (ADA, 2015).

Glycohaemoglobin test reflects average blood glucose levels over the past 3 months. This test is the most reliable test for diabetes, but it is not as sensitive as the other tests. In some individuals, it may miss pre-diabetes that could be caught by glucose tests (ADA, 2015).

Although some health care providers can use the point-of-care test, that type of measurement is not considered reliable for diagnosis. For diagnosis of pre-diabetes, the A1C test should be analysed in a laboratory. An A1C of 5.7 – 6.4% indicates pre-diabetes. An A1C of ≥6.5% is considered diabetic (ADA, 2015).

Fasting plasma glucose test measures blood glucose in people who have not eaten anything for at least 8 hours. This test is most reliable when carried out in the morning.

Pre-diabetes diagnosed using this test is known as impaired fasting glucose (IFG). Fasting glucose levels of 5.6 – 6.9 mmol/L indicate pre-diabetes, and that of ≥7 mmol/L is indicative of diabetes (ADA, 2015).

The oral glucose tolerance test (OGTT) measures blood glucose after people have not eaten for at least 8 hours and 2 hours after they drink a sweet liquid provided by the personnel taking the measurements (SEMDSA, 2017). Pre-diabetes found with this test is called impaired glucose tolerance (IGT). A blood glucose level between 7.8 – 11.0 mmol/L indicates pre-diabetes, and that above 11.0 mmol/L indicates diabetes (ADA, 2015). The recommendations for the diagnosis of diabetes are similar to those provided by the World Health Organisation (WHO, 2006).

2.4. RISK FACTORS FOR TYPE 2 DIABETES MELLITUS

T2DM is a complex attribute where common genetic variants interact with the environmental factors to modulate the risk of the disease (Murea et al., 2012). Carnethon et al. (2003) reported that family history of diabetes predisposes individuals to this condition in a large scale. Insulin resistance is a condition marked by reduced sensitivity of tissues to insulin resulting in hyperglycaemia, and is a predisposing factor for diabetes. Axelsen et al. (1999) observed that insulin resistance appears in first-degree relatives of T2DM subjects.

A wide variety of lifestyle factors such as sedentary lifestyle, smoking and alcohol consumption have a great impact on T2DM development (Hu et al., 2001). Urbanisation is associated with a more sedentary lifestyle tending to increase diabetes prevalence (Ramachandran et al, 1999), so to some extent is a proxy for lifestyle changes.

Several studies reported that T2DM arises from a combination of genetic factors and acquired factors that impair pancreatic beta-cell function; various genes such as those that encode for glucose transport are associated with beta-cell dysfunction in T2DM (Yoon and Jun, 2005; Couri et al., 2006; Trajkovski et al., 2008). Whether caused by

increased calorie intake, genetic predisposition and physical inactivity; obesity remains the major risk factor for T2DM (Singh et al., 2010).

A study by McNeely and Boyko (2004) found that people who are African-American, Asian-American, Latino-Hispanic-American, Native American, or Pacific Islander all have a greater chance of developing type 2 diabetes than other groups; although they could not definitively determine why these specific ethinicities are at increased risk for diabetes, they suspect that the risk may be associated with differences in BMI and proportion of body fat among different ethnicities. Type 2 diabetes occurs mostly in people aged 45 years and older (Centers for Disease Control, 2017). This type of diabetes is however emerging in younger people (Alberti et al., 2004).

Diet and physical fitness are the two risk factors regarded as modifiable (Jaacks et al., 2016). These two play significant roles in the reduction of many conditions other than Type 2 diabetes such as hypertension, cardiovascular diseases and obesity. They achieve their multi-disease preventing action by reducing insulin resistance which is the central condition between all these conditions; hence these two factors are crucial (Jaacks et al., 2016; Hamman, 1992). It was reported that overweight and obesity prevalence was high in primary school children from a study conducted in a rural area of Limpopo Province (Monyeki et al., 2008). It was further added that this increases the risk of developing Type 2 diabetes and other metabolic conditions for these children in adulthood (Monyeki et al., 2008).

2.5. COMPLICATIONS OF DIABETES

The acute and chronic complications of diabetes account for the morbidity and mortality associated with this disease. Acute complications include diabetic ketoacidosis, hyperosmolar hyperglycemic nonketotic coma, and hypoglycaemia (Umpierrez et al., 2002). Chronic hyperglycemia is central to the pathophysiology of chronic complications such as cardiovascular and peripheral vascular disease, retinopathy, nephropathy, and neuropathy (Chawla et al., 2016).

Long-term complications of diabetes can be classified as cardiovascular, retinopathy, renal and neural complications (Cade, 2008). These complications are thought to result from the formation of sugar and alcohols through the action of aldose reductase an enzyme found in the retina, lens, glomerulus and Schwann cell of nerves (Tang et al., 2012).

People with type 2 diabetes are two to four times more likely to develop a serious cardiovascular outcome compared with those without diabetes (Stamler et al., 1993; Huxley et al., 2006). Some large observational studies have confirmed the continuous and positive association between various measures of glycaemia (including fasting and post-load glucose levels and HbA_{1c}) and the risk of cardiovascular disease (DECODE study group, 2003; Asia Pacific Cohort Studies Collaboration, 2004).

Eye complications associated with diabetes include proliferative retinopathy, macular oedema or diabetes-related blindness (Turnbull et al., 2009). In neuropathy, sobitol is formed from glucose in nerve cells interfering with the uptake of inositol, a related sugar alcohol required for nerve signal transduction; this impairs functioning of nerves (Tang et al., 2012).

2.6. GLOBAL PREVALENCE OF TYPE 2 DIABETES MELLITUS

The incidence and prevalence rates of worldwide diabetes have doubled over the past three years. In 2010 an estimation of 285 million people worldwide had diabetes mellitus and approximately 90% of whom were diagnosed with T2DM (Danaei et al., 2011). Thus, in relation to other types of diabetes, T2DM is responsible for about 95% of diagnosed cases of diabetes in adults (Cohen et al, 2007). The global projected rise in number of people with diabetes mellitus is 439 million by 2035, which is 7.7% on the entire adult population in the world aged 20-79 years (Zimmet et al., 2001).

King et al. (1998) estimated that there would be 300 million adults with diabetes in 2025; in 2004, WHO (2004) estimated 171 million cases for year 2000 and 366 million by 2030. The incidence was already at 422 million in 2016 (WHO, 2016). Population

growth, ageing of populations, and urbanisation with associated lifestyle changes are likely to lead to a 50.7% increase in worldwide cases of diabetes by 2030 (Whiting et at, 2011)

The largest increases are expected in the older age groups in low and lower-middle income countries, with numbers more than doubling for the over 60-year age group (WHO, 2016). In high-income countries, an increase (42%) is only expected among the over 60s, with almost no change predicted for younger age groups (Whiting et al., 2011). Currently, the greatest number of people worldwide with diabetes is in the 40 – 59-year-old age group, and this is predicted to remain so in 2030, although there will be almost as many people with diabetes in the 60–79-year-old age group (Whiting et al., 2011).

The prevalence and incidence of T2DM has increased dramatically in recent years (Geiss et al., 2014). The global prevalence of T2DM rapidly increases because of lifestyle changes other than population age and urbanisation (Zimmet et al., 2001). This global burden (T2DM) is common in adult Individuals from different regions of the world and it is referred to as a major health problem.

Type 2 diabetes mellitus was relatively uncommon in developing countries decades ago, this major burden is now progressing in developed countries; hence, 80% of cases of young middle-aged individuals with diabetes mellitus worldwide occur in both developing and developed countries (Shaw et al., 2010). Family history of T2DM also has a major association with the prevalence of T2DM (Arfa et al., 2007).

With reference to the World Health Organization (WHO, 2006), diabetes affects about 347 million people on a global scale and mortality due to this condition will double between 2005 and 2030. The International Diabetes Federation (IDF, 2015) reported that 415 million adults worldwide, which represent 9% of the world, have diabetes. This condition was previously and still is common in adult people from different parts of the world and it is a major health problem (Cohen et al., 2007).

2.7. PREVALENCE OF TYPE 2 DIABETES MELLITUS IN AFRICA

Diabetes has emerged as a significant health problem in developing areas of the world; Africa included (McLarty et al, 1989). King et al. (1998) also indicated that more cases of diabetes should be expected from people residing in developing countries. According to Kengne et al. (2012) and Peer et al. (2014), type 2 diabetes accounts for greater than 90% of diabetes cases in Africa. It is further indicated that the rise in the occurrence of this condition in Africa has been principally linked to the influence by environmental factors such as transition in nutrition as well as urbanisation which is accompanied by lifestyle changes (Hu, 2011). The pathophysiology of type 2 diabetes in the African populations is still vague. This discovery is unlike in other places outside Africa where the role of genetic components on the development of type 2 diabetes is clear and understood (Kaprio et al., 1992; Lindgren et al., 2002).

In sub-Saharan Africa the rate of mortality and morbidity increases due to diabetes mellitus incidence and prevalence rate (WHO, 2004). The prevalence of diabetes in sub-Saharan Africa is variable across countries, environmental settings and ethnic groups (Kengne et al., 2012). In 2010 an approximately 12.1 million adults were diagnosed with T2DM in Africa and it has been projected to increase to 23.9 million by 2030 (Shaw et al., 2010). In rural Uganda and urban Kenya the prevalence of T2DM was at 0.6% and 12% respectively (Maher et al., 2010). A low prevalence (0 – 7%) of T2DM was reported in Cameroon, Ghana, and Nigeria. In rural Tanzania, the prevalence of adults diagnosed with T2DM ranged from 8.3 – 13.2% (Aspray et al., 2000). The incline of T2DM in Sub-Saharan Africa was also linked to the influence of environmental factors such as urbanisation and nutrition including modifiable factors such as lifestyle changes. African continent is projected to endure a major burden of diabetes mellitus in the coming decades (Guariguata et al., 2014).

Most of available data for diabetes prevalence in sub-Saharan Africa is based on reports from west, east, northeast Africa, and South Africa; little has been done in other Southern African countries. In a study that was conducted in Zimbabwe, a prevalence of > 10% was recorded (Hall et al., 2011).

2.8. PREVALENCE OF TYPE 2 DIABETES MELLITUS IN SOUTH AFRICA

According to the International Diabetes Federation (IDF, 2015) 7% of adult South Africans between the ages of 18 and 45 years have been diagnosed with diabetes. This estimates that about 3.85 million South Africans have diabetes. In 2010 the prevalence of T2DM in South Africa was estimated at 4.5%, thus a 155% incline within six years (IDF, 2009). Overall prevalence of T2DM in rural areas of South Africa is estimated at 3.9 – 8.8% (Motala et al., 2008).

Mollentze et al. (1995) has reported a prevalence of 4.8% from a study that took place in a semi-urban area of the Orange Free State province. Then Levitt et al. (1997) and Omar et al. (1993) reported 8% and 5.3% prevalence of type 2 diabetes in Cape Town and Durban respectively. These were reported years ago when the knowledge regarding type 2 diabetes was little and diagnosis was low so chances are these percentages are now higher. In the Eastern Cape, the proportion of death due to diabetes accounts for 17% of males and 23% of female deaths (Bradshaw et al., 2007).

A study conducted by Alberts et al. (2005) in a population in the Limpopo Province found the prevalence of type 2 diabetes mellitus to be 8.8 and 8.5% in women and men, respectively. Although a study conducted by Monyeki et al. (2007) found that diabetes did not exist in the paediatric population of Ellisras, associated risk factors were found to increase with time, suggesting the possibility of the emergence of type 2 diabetes in this population as age advances (Monyeki et al., 2008).

2.9. OVERVIEW OF GLUCOSE METABOLISM

Metabolism refers to the chemical reactions that take place inside the cells of living organisms which are essential for life (Marieb and Hoehn, 2014). Insulin is the most

important hormone in the metabolism of glucose. Its main effect is to lower blood glucose levels (Figure 1); nonetheless it also influences protein and fat metabolism. Circulating insulin lowers blood glucose levels in three ways. It enhances membrane transport of glucose (and other simple sugars) into most body cells, especially muscle and fat cells; inhibits the breakdown of glycogen to glucose; inhibits the conversion of amino acids or fats to glucose. These inhibiting effects counter any metabolic activity that would increase plasma levels of glucose. Elevated blood glucose levels stimulate pancreatic beta cells to secrete insulin. As body cells take up glucose and its blood levels drop, insulin secretion is suppressed (Marieb and Hoehn, 2014).

Glucagon is another hormone important in the regulation of blood glucose. Glucagon is a potent hyperglycemic agent: One molecule can cause the release of 100 million glucose molecules into the blood (Rizkalla et al., 2004). The major target of glucagon is the liver, where it promotes the breakdown of glycogen to glucose (glycogenolysis), synthesis of glucose from lactic acid and from noncarbohydrate molecules (gluconeogenesis), release of glucose to the blood by liver cells, causing blood glucose levels to rise. Its release is stimulated by falling of blood glucose levels, and suppressed by rise in blood glucose and insuling levels (Marieb and Hoehn, 2014).

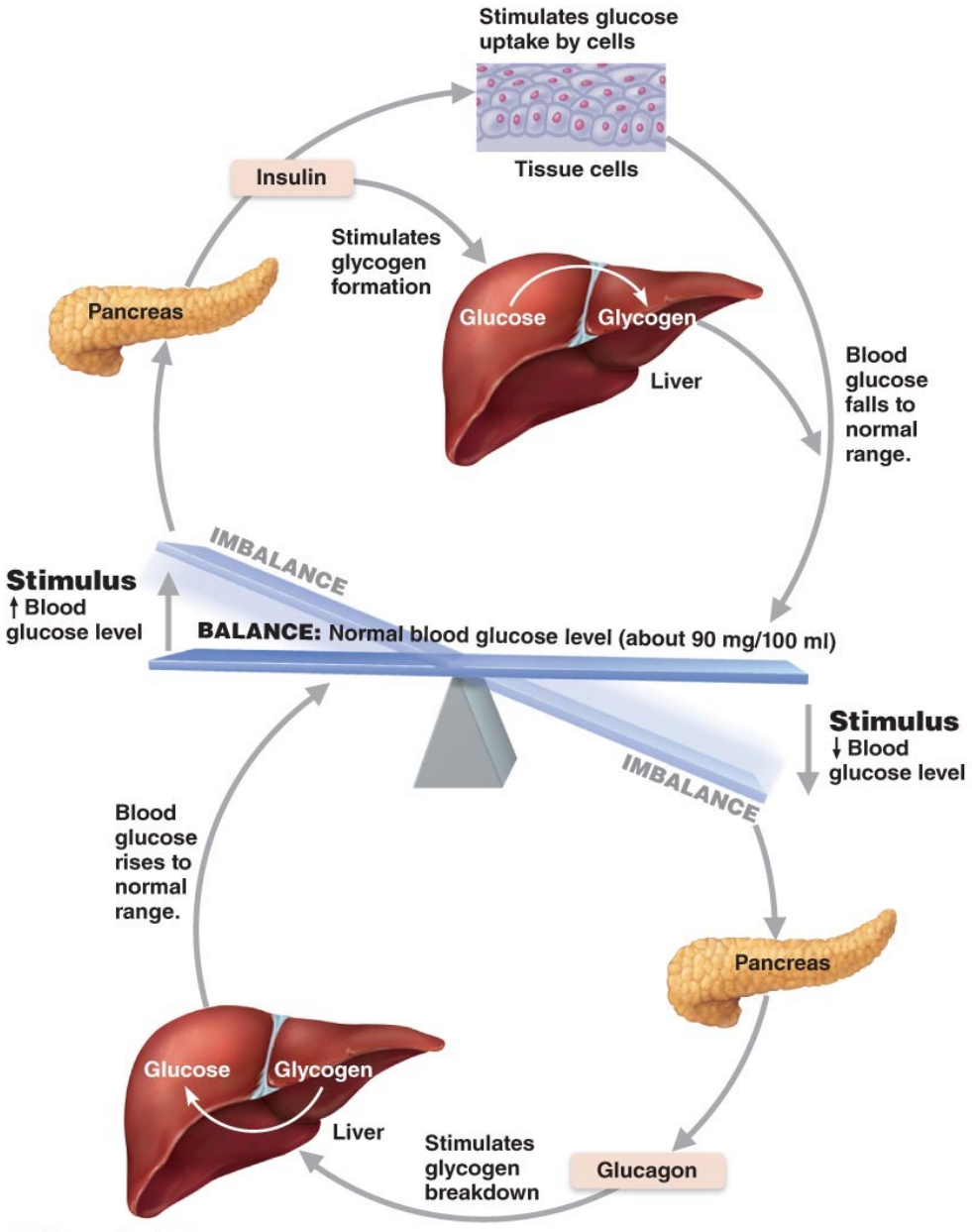

Figure 1: Insulin and glucagon from the pancreas regulate blood glucose levels (Marieb and Hoehn, 2014).

2.10. PATHOPHYSIOLOGY OF INSULIN SECRETION IN T2DM

Glucose is the principal stimulus for insulin secretion, though other stimuli such as macronutrients, hormones and neural input exist. Insulin, together with glucagon, regulates blood glucose concentrations (Kahn et al., 1997). Pancreatic β-cells secrete 0.25 – 1.5 units of insulin per hour during the fasting state, sufficient to enable glucose insulin-dependent entry into cells. This level prevents uncontrolled hydrolysis of triglycerides and limits gluconeogenesis, thereby maintaining normal fasting blood

glucose levels (Wilcox, 2005). Fasting insulin secretion accounts for over 50% of total 24 hour insulin secretion. In healthy lean individuals circulating venous (or arterial) fasting insulin concentrations are about 3 – 15 mIU/L or 18 – 90 pmol/L (Kahn et al., 1997). Meal-related insulin secretion accounts for the remaining fraction of the total daily output (Fu et al., 2013).

Type 2 diabetes mellitus results from either hyposecretion or hypoactivity of insulin (Nair, 2007). When insulin is absent, the result is type 1 diabetes mellitus; if insulin is present, but its effects are deficient, the result is type 2 diabetes mellitus (ADA, 2009). In either case, blood glucose levels remain high after a meal because glucose is unable to enter most tissue cells. High blood glucose levels rise even higher, and excess glucose begins to be lost from the body in urine (glycosuria) (Marieb and Hoen, 2014). When sugars cannot be used as cellular fuel, more fats are mobilised, resulting in high fatty acid levels in the blood, a condition called hyperlipidemia (Yang et al., 2013). In severe cases of diabetes mellitus, blood levels of fatty acids and their metabolites (acetoacetic acid, acetone, and others) rise dramatically (Hu et al., 2001). The fatty acid metabolites, collectively called ketones or ketone bodies, are organic acids. When they accumulate in the blood, the blood pH drops, resulting in ketoacidosis, and ketone bodies begin to spill into the urine (ketonuria) (Marieb and Hoehn, 2014).

2.11. PHYSICAL ACTIVITY

Physical activity is defined as any bodily movement produced by skeletal muscles that require energy expenditure (Caspersen et al., 1985). Physical activity is a key determinant of energy expenditure, and thus is fundamental to energy balance and weight control (WHO, 2004). Physical inactivity has been identified as the fourth leading risk factor for global mortality causing an estimated 3.2 million deaths globally (WHO, 2010).

Physical activity reduces risk for cardiovascular diseases and diabetes and has substantial benefits for many conditions, not only those associated with obesity.

Physical activity has been shown to reduce the risk of conditions that often co-exist with diabetes (Piva et al., 2015). For example, physical activity reduces blood pressure, improves the level of high density lipoprotein cholesterol, and improves control of blood glucose in overweight people, even without significant weight loss (WHO, 2004).

For physical activity, it is recommended that individuals engage in adequate levels throughout their lives. Different types and amounts of physical activity are required for different health outcomes: at least 30 minutes of regular, moderate-intensity physical activity on most days reduces the risk of cardiovascular disease and diabetes, colon cancer and breast cancer (WHO, 2010).

Physical activity has been recognised as an important tool for prevention of diseases in developed countries (Bassuk and Manson, 2003; Haskell, 2003; McKechnie and Mosca, 2003). Research clearly shows that physical inactivity is associated with an increased risk of chronic diseases (Oguma and Shinoda-Tagawa, 2004), while increasing regular PA has been presumed to be an important tool for the prevention of diseases (U.S. Department of Health and Human Services, 1996).

Globally in 2010, around 23% of adults aged 18+ years were insufficiently physically active; with women even less active than men (WHO, 2010). It is suggested that physically inactive children are likely to become inactive adults (Benefice, 1993). As a result, these children have the potential to become extensive consumers of health care, contributing significantly to health care costs in the future (Riddoch and Boreham, 1995). The prevention of chronic diseases should therefore begin in childhood (Berenson and Pickoff, 1995; Aarts et al., 1997).

2.12. STATE OF PHYSICAL ACTIVITY IN SOUTH AFRICA

The prevalence of physical inactivity, defined as doing no or very little physical activity at work, at home, for transport or during leisure time, has been estimated to be 43% – 49% in South Africans 15 years of age and older (Joubert et al., 2007). Micklesfield et

al. (2014) found that PA level is low among South African children, and correlated it with factors such as Socio-Economic Status (SES), maternal, household and community level. As children grow old, their level of PA decreases (Mokabane et al., 2014), this shows that the level of PA is expected to decrease further with time.

Black South African women were identified having the country's highest levels of inactivity, overweight and obesity (Kolbe-Alexander et al., 2012). A WHO survey found that less than one third of South Africans met the American College of Sports Medicine and Centers for Disease Control's recommendations for health-enhancing physical activity (to accumulate 30 minutes of moderate activity on most, but preferably all days of the week), and that nearly half (46%) were reportedly inactive (Kolbe-Alexander et al., 2012). Measures of physical activity in rural-dwelling South African men and women confirmed that majority of PA in this population is through walking (Micklesfield et al., 2014).

The low level of PA in relation to SES, household and community educational level provides more reason for why it is important to understand factors, specific to rural South African youth, associated with physical activity.

2.13. ASSOCIATION OF PHYSICAL ACTIVITY WITH PLASMA GLUCOSE LEVELS AND INSULIN

Physical activity (PA) plays an important role in the control of both weight and plasma glucose, thus reducing the likelihood of developing T2DM (Sanz et al., 2010). It has been proven that PA improves insulin sensitivity, which helps with the uptake of glucose by cells (Sanz et al., 2010). Buman et al. (2014) found that reallocating 30 minutes per day of sedentary time to light-intensity PA was associated with 2.4% reduction in insulin levels. Physical activity promotes the treatment of insulin resistance through a reduction in white adipose tissue (WAT) mass (Ross and Bradshaw, 2009). Accumulation of WAT results in the reduction in the phosphorylation of insulin receptors in adipocytes, thereby, decreasing the expression of insulin receptor, subsequently leading to lower glucose uptake into adipocytes (Algenstaedt et al., 2004; Buren et al., 2003). Physical activity also lowers levels of glycohaemoglobin, which is a marker for T2DM (Dunstan et al., 2002). Information about the benefits of PA on the prevention and treatment of T2DM is scanty in rural South African populations.

2.14. SUMMARY

It is evident that diabetes mellitus is a growing concern both globally and in South Africa. The prevalence and incidence of T2DM has increased dramatically in recent years (Geiss et al., 2014) and continues to do so because of lifestyle changes other than population age and urbanisation (Zimmet et al., 2001). In 2015, the International Diabetes Federation (IDF, 2015) reported that 7% of adult South Africans between the ages of 18 and 45 years have been diagnosed with diabetes

A wide variety of lifestyle factors such as PA, sedentary lifestyle, smoking and alcohol consumption have a great impact on T2DM development (Hu et al., 2001). The biggest culprit, obesity (Singh et al., 2010), is a modifiable risk factor and knowledge of lifestyle adjustments that can curb this epidemic is essential among rural South African populations.

Complications associated with type 2 diabetes can be devastating and life-threatening. It is important that effective diagnostic procedures are employed for proper management of this disease. Fasting glucose and 2-hour OGTT are shown to be effective in this regard (ADA, 2015). The level of PA in South Africa, especially in children and adolescents, is concerningly low and continues to decrease (Joubert et al., 2007; Mokabane et al., 2014), indicating a need for intervention strategies. The association of PA with SES, household and community educational level opens a window for further research on the impact that the environment has on the level of PA of a population.

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CHAPTER 3

MATERIALS AND METHODS

3.1. Geographical area

3.2. Sample

3.3. Sampling and data collection

3.4. Anthropometry

3.5. Physical activity

3.6. Fasting blood samples

3.7. Employment and family history of diabetes

3.8. Quality control

3.9. Statistical analysis

3.10. References

3.1. GEOGRAPHICAL AREA

The participants of the study were young adults (aged 22 – 30 years) residing in 42 rural villages situated in Lephalale (previously known as Ellisras), Limpopo Province. These villages are situated approximately 70km from the Lephalale town (23^0 40S 27^0 44E) and are adjacent to the Botswana border. Major sources of employment are Matimba power station, Grootegeluk coal mine and education. Youth unemployment remains high to this age (36.8%) (Statistics South Africa, 2017). Poverty and low life expectancy are common in rural South African population, Ellisras rural area included (Bradshaw & Steyn, 2001).

Figure 2: Map showing Ellisras area (Google Maps).

3.2. SAMPLE

Details of the ELS research design and sampling have been reported elsewhere (Monyeki et al., 2000). For the purpose of this analysis, a portion of the ELS sample was recruited. In total, 717 young adults (352 males and 365 females), aged 18 to 28 years, who are part of the ELS took part in the survey.

The Turfloop Research Ethics Committee of the University of Limpopo, granted ethical approval prior to the survey (Project Identification ID: TREC/P/356/2017: PG). Permission was granted by tribal authorities and the principals of schools. Participants read and signed informed consent.

3.4. ANTHROPOMETRY

All participants underwent a series of anthropometric measurements. Height was measured to the nearest 0.1 cm using a standardised Martin anthropometer. Waist circumference (WC) was measured using a flexible steel tape at minimal inspiration to the nearest 0.1 cm, midway between the last rib and the iliac crest, with the participants standing. Waist-to-height ratio (WHtR) was calculated as WC divided by height. These anthropometric measurements were undertaken by trained personnel and were done according to the International Society for the Advancement of Kinanthropometry (ISAK) (Norton and Olds, 1996). Data form for the anthropometric variables is attached as Appendix A.

3.5. PHYSICAL ACTIVITY

The International Physical Activity Questionnaire (IPAQ) was found to be reliable and valid for assessment of PA (Craig et al., 2003). The questionnaire was used to record data regarding work, leisure, and travel activities both on week days and weekends. The score for these activities were attained by multiplication of the number of reported activities with the number of days engaged in those activities per week. The total PA score was calculated as a summation of the types of activities for each participant (IPAQ, 2005). The duration of these periods was added up to give a mean duration for

moderate to vigorous physical activities during weekdays and weekends. Metabolic equivalents (METs) express the energy cost of physical activities as a multiple of the resting metabolic rate and yield a score in MET-minutes. MET-minutes were obtained by multiplying the MET score (8 for vigorous and 4 for moderate activity) by the minutes performed (Ainsworth et al., 2000).

The principal investigator with the help of Ellisras local teachers translated the questionnaire from English to the two locally spoken languages (Northern Sotho and Setswana) and then translated back to English. The back translation to English showed no disparity with the Northern Sotho and Tswana languages.

The Senior Northern Sotho and Tswana speaking Physiology and Environmental Health department students of the University of Limpopo who were specifically trained for using this questionnaire, interviewed the participants at local schools/halls; with each interview lasting for about 30 min. The questionnaire is attached as Appendix D.

3.6. FASTING BLOOD SAMPLES

Participants were asked to fast for 8–10 hours before blood collection. All collections were done by a registered nurse at local schools/halls. Fasting venous blood samples were collected in the morning (Kemper, 2004). Fasting blood samples were collected into 4 mL grey and gold top vacutainer tubes (vacutainer BD^{TM}) containing sodium fluoride and gel, respectively. Samples were then placed in a cooler box with ice (0–8 $^{\circ}$C) on site. At the laboratory, fasting blood samples were centrifuged to obtain serum at 2500 revolutions per minute (rpm) for 15 minutes. Clotted and hemolysed samples were discarded. Serum was stored at −80 $^{\circ}$C for later analysis. Glucose and insulin were measured on AU480 autoanalyser and Access 2 immunoassay, respectively, using reagents supplied by Beckman Coulter USA. All measurements were done at the University of Limpopo's Medical Sciences laboratory by trained medical science laboratory technicians and physiology postgraduate students.

Figure 3: Picture showing field workers analysing blood samples.

3.7. EMPLOYMENT AND FAMILY HISTORY OF DIABETES

Information was collected regarding employment and family history of diabetes by means of a questionnaire. Participants were asked to answer ‘yes’ if they are employed and ‘no’ if not. Participants also had to answer ‘yes’ if anybody in their family had or ever had diabetes and ‘no’ if not. The questionnaire is attached as Appendix D.

3.8. QUALITY CONTROL

All training of anthropometric measurements was done in accordance with the standard procedures of the ISAK (Norton and Olds, 1996). Reliability and validity of anthropometric measurements were reported elsewhere (Monyeki et al., 2002). Personnel were trained to have technical error of the measurement (TEM) for anthropometric variables that are within acceptable standards as per the ISAK. In brief, the absolute and relative values for intra- and inter-tester technical error of measurements (% TEM) for stature, ranged from 0.04-4.16 cm (0.2-5.01%) and waist

circumference measurements ranged from 0.0-3.4 cm (0-4%) (Monyeki et al., 2002; 2008).

3.9. STATISTICAL ANALYSIS

Descriptive statistics

Descriptive statistics of the population characteristics, anthropometric measurements and blood parameters were reported by gender. All participants were classified as inactive, minimally active, or sufficiently active according to the IPAQ cut-off point that categorises physical activity as inactive (<600 MET-min/week), moderately activity (600–1500 MET-min/week) and sufficiently active (≥1500 MET-min/week) (IPAQ, 2005).

Prevalences

The American Diabetes Association (ADA) cut-off points were used to define pre-diabetes as plasma glucose level between 5.6 and 6.9 mmol/L (inclusive), diabetes as plasma glucose level ≥7.0 mmol/L and insulin resistance as a HOMA-IR score ≥2.5 (ADA, 2016). A high WC was defined as WC ≥80cm for females and WC ≥94cm for males (IDF, 2006). An internationally proposed cut-off for WHtR of 0.5 was used (Ashwell, 2005).

Figure 4: Picture shows field workers capturing blood analysis results.

Linear regression

A linear regression model was used for assessing the relationship between physical activity levels and plasma glucose, insulin and obesity indices (BMI, WC and WHtR) both unadjusted and adjusted for family history of diabetes, age, gender and dietary intake.

Logistic regression

Logistic regression was used to assess the odds of having diabetes with low PA and obesity for unadjusted and adjusted for family history of diabetes, age, gender and dietary intake.

Statistical software and p-value

All the statistical analyses were done using SPSS software version 23 (IBM, Armonk, NY, USA). Statistical significance was set at $p < 0.05$.

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CHAPTER 4

RESULTS AND DISCUSSION

4.1. Results and discussion

4.2. References

4.1 RESULTS AND DISCUSSION

The aim of this study was to assess the relationship between PA and the risk of having type 2 diabetes mellitus in Ellisras rural young adults. This study is the first to examine the independent association of PA with T2DM and IR, taking into account obesity indices, in the Ellisras Longitudinal Study population.

Table 1 shows the descriptive statistics for age, BMI, PA, WC, WHtR, glucose and HOMA-IR. Males had significantly ($p<0.05$) lower WC and WHtR compared to females. The levels of glucose and HOMA-IR were significantly lower in males (5.56 mmol/L; 2.10) compared to females (5.75 mmol/L; 2.89), and the level of PA was low in females (538.65 MET-min/week) compared to males (670.34 MET-min/week).

Table 1: Descriptive statistics for physical activity in MET-minutes per week and other population characteristics associated with type 2 diabetes mellitus in 670 ELS young adults (males n= 332; females n= 338).

Risk factor	Male (Mean±SD)	Female (Mean±SD)	Total (Mean±SD)
Age	23.47±1.92	23.77±1.96	23.62±1.95
BMI (Kg/m^2)	21.26±3.63	25.76±6.11	23.5±5.51
Waist circumference (cm)	74.90±9.56*	82.50±14.30*	78.72±12.75
Waist-to-height ratio	0.44±0.12*	0.51±0.10*	0.47±0.11
Glucose (mmol/L)	5.56±0.93	5.75±1.54	5.66±12.81
HOMA-IR	2.10±1.86*	2.89±4.04*	2.49±3.53
Physical activity (MET-minutes per week)	670.34±969.68	538.65±896.09	603.90±934.91

* = $p<0.05$. HOMA-IR: Homeostatic model assessment for insulin resistance, calculated as: (fasting insulin [µU/mL] x fasting glucose [mmol/L])/ 22.5. METs are multiples of the resting metabolic rate and

yield a score in MET-minutes, which is computed by multiplying the MET score (eight for vigorous and four for metabolic activity and travel related walking/cycling) by the minutes performed. *p<0.05

Table 2 shows descriptive statistics for MET-min per week in the work, leisure and travel domains and the proportions of total physical activity with the corresponding plasma glucose concentrations of each age group. Males (an average of 783.05 MET-min per week) were significantly ($p < 0.05$) more active than females (an average of 777.52 MET-min per week). These findings corroborate those of Micklesfield et al. (2014) that suggest that females are less active than males, and the overall PA level is low in a rural population of South Africa. It was further observed that males spend generally more time doing work-related activities than females. According to Puoane et al. (2007), the low levels of PA in rural communities may be attributed to factors such as poverty, crime, lack of security, time constraints, inability to afford exercise equipment and lack of recreational facilities.

The overall plasma glucose concentrations for females (average 5.67 mmol/L) were significantly higher than those of males (average 5.51 mmol/L). This is similar to what McCollum et al. (2005) found, although their study was based on an older population, where women were significantly older than men, and had less education and lower incomes (which may lead to the consumption of unbalanced diets).

Table 2: Descriptive statistics for MET-min (metabolic equivalent minutes) per week in work, leisure and travel domains and proportions of total physical activity with corresponding plasma glucose concentrations of each age group for 717 ELS young adults aged 18 to 28 years

	Males (n = 352)						Females (n = 365)					
Age (Mean ± SD)	**n**	**Mean (SD) MET-min/week**				**Plasma Glucose Concentration (mmol/L)**	**n**	**Mean (SD) MET-min/week**				**Plasma Glucose Concentration (mmol/L)**
		Work	**Travel**	**Leisure**	**Total**			**Work**	**Travel**	**Leisure**	**Total**	
20.99 ± 0.7	92	40.00 * (69.66)	480.44 (153.06)	235.83 (93.18)	756.27 (276.13)	5.57 (0.97)	77	31.17 * (63.78)	457.77 (183.89)	265.86 (492.07)	754.80 (300.91)	5.50 (0.88)
23.07 ± 0.6	104	58.46 * (77.42)	495.22 (190.56)	258.20 (151.06)	811.88 (299.22)	5.63 (1.005)	107	55.33 * (76.46)	556.91 (380.53)	267.15 (422.79)	879.39 (478.12)	5.70 (0.867)
24.95 ± 0.6	109	57.25 (77.05)	485.36 (175.88)	261.27 (104.61)	803.88 (305.65)	5.49 (0.94)	128	45.00 (72.22)	468.38 (157.14)	207.80 (522.76)	721.18 (235.28)	5.69 (2.19)
26.61 ± 0.4	47	68.09 (79.96)	430.98 (156.15)	261.1 (132.15)	760.17 (264.38)	5.34 (0.737)	53	57.36 (77.46)	439.85 (154.72)	257.51 (568.56)	754.72 (272.55)	5.77 (0.89)

METs are multiples of the resting metabolic rate and yield a score in MET-minutes, which is computed by multiplying the MET score (8 for vigorous and 4 for metabolic activity and travel related walking/cycling) by the minutes performed. * $p < 0.05$.

Table 3 presents the prevalence of unemployment, family history of diabetes, physical inactivity, high waist circumference, high waist-to-height ratio, insulin resistance, pre-diabetes and diabetes in Ellisras young adults. The prevalence of physical activity inactivity (63.9% males; 67.7% females) is high in this population as defined by the international physical activity questionnaire (IPAQ). This study supports an assertion by Amosun et al. (2007) that over a third of South African youth are inactive (67.3% males and 71.0% females in this study).

The prevalence of overweight and obesity (BMI > 25kg/m^2) (11.9% males; 49.4% females) was higher than the 28.6% reported by the Transition and Health during Urbanisation in South Africa (THUSA) study conducted on black South Africans residing in the North West province (Kruger et al., 2002), although the study only focused on women. Another study conducted in a different rural population in the Limpopo Province found slightly higher (44%) prevalence of obesity (Alberts et al., 2005).

The prevalence of central obesity (WC > 80cm and WC > 94cm for females and males respectively; WHtR > 0.5) in the ELS study population was markedly higher (high WC 4.2% males, 51.2% females; high WHtR 12.3% males, 48.5% females) than that (by WC 15.95%; by WHtR 3.2%) reported by Alberts et al. (2005) in another Limpopo Province rural population. Variation in factors such as infectious disease burden and social influences may account for the difference among populations (Kimani-Maruge et al., 2010).

Insulin resistance (HOMA-IR score ≥ 2.5) prevalence was found to be 22.9% in males and 29.3% in females. This prevalence is higher than that reported by Mamabolo et al. (2007) in another South African population. This is in contradiction with the results by Phanzu et al. (2014) which found a prevalence of 44.8% in a Congolese population. Insulin resistance has been shown to be increased in populations with high levels of overweight and obesity (Mamabolo et al., 2007); this may explain the difference in prevalence among different populations.

In the current study the prevalence of pre-diabetes was high (males = 38.6%; females = 43.5%) compared to the 25% (for both genders each) found by Okafor (2012) in rural Africans living in Nigeria. Type 2 diabetes mellitus (defined as fasting plasma glucose level ≥7.0 mmol/L) was found in 9.6% of males and 10.1% of females. This prevalence is higher than what Alberts et al. (2005) found in a different Limpopo Province rural population (8.5% males and 8.8% females). This prevalence is also higher than the global estimate which was set to be 7.7% by 2030 (Shaw et al., 2010). This high prevalence could be due to the high levels of inactivity (males = 63.9%; females = 67.7%) and high family history of diabetes (males = 21.6%; females = 28.5%) in this population. The high prevalence and long-term implications on health make diabetes a major concern for developing countries (Engelgau et al., 2004).

Table 3: Prevalence of unemployment, family history of diabetes, physical inactivity, high waist circumference, high waist-to-height ratio, insulin resistance, pre-diabetes and diabetes.

Variables	Males %(n)	Females %(n)	Total %(n)
Unemployment	65.3(230)	70.7(258)	68(488)
Family history of diabetes	21.6(76)	28.5(104)	25.1(180)
Physical inactivity	63.9(225)*	67.7(247)*	65.8(472)
BMI > 25kg/m^2	11.9(40)*	49.4(167)*	30.8(207)
High waist circumference	4.2(14)*	51.2(173)*	27.7(187)
High waist-to-height ratio	12.3(41)*	48.5(164)*	30.4(205)
Insulin resistance	22.9(76)*	29.3(99)*	26.1(175)
Pre-diabetes	38.6(128)*	43.5(147)*	41.1(275)
Diabetes	9.6(32)*	10.1(34)*	9.85(66)

*p<0.05

Table 4 shows the prevalence of IR (HOMA-IR ≥2.5) and diabetes (glucose ≥7.0mmol/L) stratified by the level of PA. Those in the inactive category (<600 MET-min/week) had the highest prevalence of IR (38.1%) and diabetes (13.6%), and those in the sufficiently active category (≥1500 MET-min/week) had the lowest prevalence of IR (17.4%) and diabetes (6.6%).

Table 4: Prevalence of IR and T2DM stratified by PA.

PA level		HOMA-IR		Glucose
Inactive (<600 MET-min/week)	<2.5	61.90%	<7.0	86.4%
	≥2.5	**38.10%**	≥7.0	**13.60%**
Minimally active (600 - 1500 MET-min/week)	<2.5	77.00%	<7.0	90.7%
	≥2.5	**23.00%**	≥7.0	**9.30%**
Sufficiently active (≥1500 MET-min/week)	<2.5	82.60%	<7.0	93.40%
	≥2.5	**17.40%**	≥7.0	**6.60%**

METs are multiples of the resting metabolic rate and yield a score in MET-minutes, which is computed by multiplying the MET score (eight for vigorous and four for metabolic activity and travel related walking/cycling) by the minutes performed.

Table 5 shows linear regression coefficient for the relationship between physical activity (mean MET-min/week), anthropometric (WC and WHtR) and biochemical variables (glucose and insulin). A significant relationship ($p<0.05$) was observed between PA and glucose (ß= 5.481; 95%CI= 5.368 – 5.596), WC (ß= 78.573; 95%CI= 77.387 – 79.759), and WHtR (ß= 0.476; 95%CI= 0.465 – 0.486) for both unadjusted and adjusted for age, gender, family history of diabetes, BMI, WC, WHtR, carbohydrate intake and total fat intake. This shows that the least active subjects were also the most obese, without adjusting for age, gender, family history of diabetes, and diet; however, after adjusting, the correlation was no longer significant. Similar results were found by others (Kruger et al., 2002; Paeratakul et al., 1998).

The association between PA and insulin resistance was found to be significant, this substantiates the findings of Plasqui and Westerterp (2007).

Table 5: Linear regression coefficient for the relationship between physical activity (mean MET-min/week); glucose, insulin, waist-to-height ratio and waist circumference.

	Unadjusted				Adjusted for age, gender, family history of diabetes, BMI, WC, WHtR, carbohydrate intake, total fat intake.			
	Beta	p-value	95% confidence interval		Beta	p-value	95% confidence interval	
			Lower	Upper			Lower	Upper
Glucose	5.481	0.000**	5.368	5.594	5.715	0.000**	4.545	6.885
Insulin	8.710	0.000**	7.614	9.807	-1.793	0.755	-13.088	9.501
WHtR	0.476	0.000**	0.465	0.486	0.192	0.000**	0.087	0.296
WC	78.573	0.000**	77.387	79.759	37.572	0.000**	25.970	49.174

* = p<0.05; ** = p<0.01

Table 6 shows logistic regression for the relationship between low PA and diabetes, insulin resistance, high WC and high WHtR. A significant relationship (p<0.05) was found between PA and diabetes (ß= 2.702; 95%CI= 1.616 – 4.518) and insulin resistance (ß= 1.755; 95%CI= 1.226 – 2.513), both unadjusted and adjusted for age, gender, family history of diabetes, BMI, WC, WHtR, carbohydrate intake and total fat intake. This supports an assertion by Sanz et al. (2010) that PA should be part of any therapeutic strategy to slow the development of T2DM.

Table 6: Logistic regression coefficient for the relationship between low physical activity; diabetes, insulin resistance, high waist-to-height ratio and high waist circumference.

* = p<0.05; ** =p<0

	Unadjusted				Adjusted for age, gender, family history of diabetes, BMI, WC, WHtR, carbohydrate intake, total fat intake.			
	Beta [Exp(B)]	p-value	95% confidence interval		Beta	p-value	95% confidence interval	
			Lower	Upper			Lower	Upper
Diabetes	2.702	0.000**	1.616	4.518	2.890	0.000**	1.715	4.870
Insulin resistance	1.755	0.002**	1.226	2.513	1.819	0.001**	1.266	2.614
High WHtR	0.904	0.584	0.629	1.299	1.018	0.930	0.677	1.532
High WC	0.766	0.166	0.524	1.117	0.884	0.600	0.557	1.402

Parts of the limitations of the study include a low response rate as only a portion of the residents of Limpopo Province were involved. Hence caution should be taken not to generalise the results for rural South African populations. This study is cross-sectional thus, longitudinal data needs to be collected in order to conclude on the causal relationship between PA and T2DM in this population (Kemper, 2004). Moreover, T2DM is a complex disease with numerous risk factors; PA alone is not enough to predict the possibility of developing T2DM (Amosun et al., 2007), as reported in other studies (Amosun et al., 2007; Marcato-Mchunu and le Roux, 2010). However, PA plays an important role in the control of both body weight and plasma glucose, thus reducing the likelihood of developing T2DM (Sanz et al., 2010). Recall of the time spent doing physical activity could introduce bias in the study, particularly in Sub-Saharan Africa, given the illiteracy level (Monyeki et al., 2007). However, well-trained fieldworkers were reported to provide accurate information in South Africa (Monyeki et al., 2013) as was the case in the current study.

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CHAPTER 5

INTRODUCTION, SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

5.1. Introduction

5.2. Summary

5.3. Conclusion

5.4. Recommendations

5.5. References

5.1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is the most common form of diabetes and its prevalence is increasing both locally and globally with estimates of it reaching about 592 million world-wide by the year 2035 (Bartoli et al. 2011; Guariguata et al., 2014). Some of the risk factors of T2DM are pre-diabetes, insulin resistance and obesity (Guariguata et al, 2014). These risk factors are reported to commence at an early age and progresses into adulthood. As the prevalence of diabetes continues to increase and prevention of T2DM becomes a worldwide concern, there is convincing evidence that T2DM can be prevented or delayed through lifestyle modifications which include change in diet, increased physical activity and maintaining normal body weight (Gillies et al., 2007; Knowler et al., 2002). Physical activity has been reported to play a major role in the control of chronic diseases of lifestyle, including diabetes (Sanz et al., 2010). Little is known about the effect of habitual physical activity on the development of T2DM and its associated risk factors in rural African populations.

5.2. SUMMARY

Chapter 1 addressed the need for studying the relationship between physical activity and type 2 diabetes mellitus in rural young adults in Ellisras area in Limpopo Province, South Africa. The need for assessing the state of physical activity and prevalence of type 2 diabetes mellitus and its associated risk factors in the Ellisras rural young adults was indicated. In order to answer the above mentioned statements successfully, the following objectives of the study were outlined in the first chapter:

i. investigate the prevalence of physical inactivity among ELS young adults aged 22 to 30 years.
ii. investigate the prevalence insulin resistance among ELS young adults aged 22 to 30 years.
iii. investigate the prevalence T2DM among ELS young adults aged 22 to 30 years.
iv. assess the prevalence of overweight and obesity using a variety of anthropometric indeses among ELS young adults aged 22 to 30 years.

v. assess the relationship between PA and levels of glucose among ELS young adults aged 22 to 30 years.
vi. assess the relationship between PA and insulin resistance, obesity indices and T2DM among ELS young adults aged 22 to 30 years.
vii. determine the risk of having T2DM with low levels of PA.

In chapter 2, literature on the topic was reviewed. We reviewed the literature on the burden of diabetes mellitus both globally and in South Africa, the factors contributing to the increasing prevalence, the different diagnostic procedures as well as the role of physical activity in the control of diabetes and its risk factors (Whiting et al, 2011; Geiss et al, 2014; Danaei et al., 2011; Kengne et al., 2012; Guariguata et al., 2014; International Diabetes Federation, 2015; Motala et al., 2008).

Chapter 3 indicated the methodology and the statistical analysis of the data collected. Linear regression was used to determine the association between physical activity and type 2 diabetes mellitus among Ellisras rural young adults. Logistic regression was used to determine the risk of having diabetes with low physical activity levels.

Chapter 4 described the results and discussion of the study. There was significant ($P<0.05$) association between physical activity with type 2 diabetes mellitus, insulin resistance and obesity indices among Ellisars rural young adults aged 22 to 30 years. Therefore, the present study concurs with the previous studies on the association of physical activity with type 2 diabetes mellitus (Okafor, 2012; Plasqui and Westerterp, 2007; Sanz et al., 2010).

In chapter 5, a summary overview of the dissertation was presented together with conclusions and recommendations of the study which will help create a data set to aid with lifestyle intervention programmes to treat and prevent further development of diabetes among a rural population in South Africa.

5.3. CONCLUSIONS

The conclusions of the study are provided in relation to the objectives and hypothesis set out in chapter 1.

Objective 1: To investigate the prevalence of physical inactivity among Ellisras Longitudinal Study among young adults aged 22 to 30 years.

Hypothesis 1: The prevalence of physical inactivity is similar to those studied previously in South Africa.

The results of this study show that the level of physical inactivity is high (65.8%) in this population as defined by the IPAQ, with females even less active than males. This is similar to results from previous studies conducted in South Africa (Micklesfield et al., 2014; Joubert et al., 2007; Kolbe-Alexander et al., 2012). According to Puoane et al. (2007), the low levels of PA in rural communities may be attributed to factors such as crime, inability to afford exercise equipment and lack of recreational facilities.

In light of the above findings, hypothesis 1 was therefore accepted.

Objective 2: To investigate the prevalence of insulin resistance among Ellisras Longitudinal Study young adults aged 22 to 30 years.

Hypothesis 2: The prevalence of insulin resistance is similar to those studied in other parts of the world.

Insulin resistance (HOMA-IR score ≥ 2.5) prevalence was found to be 22.9% in males and 29.3% in females. The prevalence in this population is high, as it has been shown that low levels of PA are associated with high incidences of IR (Plasqui and Westeterp, 2007; Ross and Bradshaw, 2009). This prevalence is higher than that reported by Mamabolo et al. (2007) in another South African population.

In light of the above findings, hypothesis 2 was therefore partially accepted.

Objective 3: To investigate the prevalence of type 2 diabetes mellitus among Ellisras Longitudinal Study young adults aged 22 to 30 years.

Hypothesis 3: The prevalence of type 2 diabetes mellitus was similar to those studied in other parts of the world.

Type 2 diabetes mellitus (defined as plasma glucose level ≥7.0 mmol/L) was found in 9.6% of males and 10.1% of females. The prevalence is high in this population following the high prevalence of inactivity and insulin resistance. The high prevalence is expected as urbanisation and modernisation continue to increase in South Africa (Cohen, 2007). This prevalence is higher than what Alberts et al. (2005) found in a different Limpopo Province rural population (8.5% males and 8.8% females). This prevalence is also higher than the global estimate which was set to be 7.7% by 2030 (Shaw et al., 2010).

In light of the above findings, hypothesis 3 was therefore partially accepted.

Objective 4: To assess the prevalence of overweight and obesity using a variety of anthropometric indeses among ELS young adults aged 22 to 30 years.

Hypothesis 4: The prevalence of overweight and obesity is high in this population, especially in females.

The prevalence of overweight and obesity (BMI > 25kg/m^2) was higher (30.8%) than the 28.6% reported by the Transition and Health during Urbanisation in South Africa

(THUSA) study conducted on black South Africans residing in the North West province (Kruger et al., 2002). The prevalence of central obesity (WC > 80cm and WC > 94cm for females and males respectively; WHtR > 0.5) in the ELS study population was markedly higher (by WC 27.7%; by WHtR 30.4%) than that (by WC 15.95%; by WHtR 3.2%) reported by Alberts et al. (2005).

In light of the findings above, hypothesis 4 was therefore accepted.

Objective 5: To assess the relationship between PA and levels of glucose among ELS young adults aged 22 to 30 years.

Hypothesis 5: Physical activity is significantly associated with levels of glucose.

A significant relationship ($p<0.05$) was observed between PA and glucose (ß= 5.481; 95%CI= 5.368 – 5.596), for both unadjusted and adjusted for age, gender, family history of diabetes, BMI, WC, WHtR, carbohydrate intake and total fat intake. Individuals categorised as inactive (<600 MET-min/week) had the highest prevalence of elevated glucose levels (13.6%), and those categorised as sufficiently active (≥1500 MET-min/week) had the lowest prevalence of elevated glucose levels (6.6%).

In light of the findings above, hypothesis 5 was therefore accepted.

Objective 6: To assess the relationship between physical activity and insulin resistance, obesity indices and type 2 diabetes mellitus among Ellisras Longitudinal Study young adults aged 22 to 30 years.

Hypothesis 6: Physical activity will be significantly associated with insulin resistance, obesity indices and type 2 diabetes mellitus.

Linear regression found a significant correlation between physical activity and indices of obesity; this shows that the least active subjects were also the most obese, without adjusting for age, gender, family history of diabetes, and diet; however, after adjusting, the correlation was no longer significant.

The association between physical activity and insulin resistance was found to be significant, this is similar to what other researchers found (Plasqui and Westerterp, 2007). A significant relationship ($p<0.05$) was found between PA and diabetes (ß= 2.702; 95%CI= 1.616 – 4.518) both unadjusted and adjusted for age, gender, family history of diabetes, BMI, WC, WHtR, carbohydrate intake and total fat intake.

In light of the findings above, hypothesis 6 was therefore partially accepted.

Objective 7: To determine the risk of having T2DM with low levels of PA.

Hypothesis 7: The risk of having T2DM will increase with a decreasing level of PA.

Logistic regression showed a significant relationship between physical activity and type 2 diabetes mellitus, both unadjusted and adjusted for age, gender, family history of diabetes, and diet. This shows that low levels of PA increase the odds of having T2DM in the ELS population. An assertion by Sanz et al. (2010) that PA should be part of any therapeutic strategy to slow the development of T2DM is supported.

In light of the findings above, hypothesis 7 was therefore partially accepted.

5.4. RECOMMENDATIONS

We recommend that:

i. A longitudinal assessment of the relationship between physical activity and type 2 diabetes mellitus be conducted to determine the trends over time in South African rural young adults.

ii. An intervention study be conducted to assess the acute effects of different types and intensities of physical activity on blood glucose levels.

iii. Physical activity be encouraged in this population for both management of anthropometric indices as well as diabetes mellitus.

iv. Early intervention and management of risk factors for diabetes mellitus in Ellisras rural children could benefit the growing rural South African population as CVDs risk factors associated with type 2 diabetes mellitus will be combated at an early age.

v. More biochemical parameters should be included in the ELS.

- To investigate the development of biological and behavioural risk factors for CVDs and/or diabetes in rural South African population over time.

- To investigate the changes that occur in serum levels of a variety of biochemical parameters related to CVDs, obesity and diabetes in this rural South African population over time.

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APPENDICES

APPENDIX A (DATA FORM)

Subject Name:			
Subject Number:			
Code of the Fieldworker: ……			
Observation Date:			
Name Of School/Village:……			
School Number/Village Number: ……			
Last Grade at school ……..			
Gender:	Male 1	Female 2	
Date of Birth:			
Weight (Kg):			
Height (cm):			

Waist (cm):
Plasma Glucose (mmol/L):

APPENDIX B (CONSENT FORM)

Project title: **The relationship between physical activity and the risk of type 2 diabetes mellitus in Ellisras rural young adults aged 22 to 30 years: Ellisras Longitudinal Study**

Project leader: **Prof K.D. Monyeki**

Researcher: **Miss M. Matshipi**

I, hereby voluntarily consent to participate in the following project: “The relationship between physical activity and the risk of type 2 diabetes mellitus in Ellisras rural young adults aged 22 to 30 years: Ellisras Longitudinal Study.”

I understand that:

1. My responses will be treated with confidentiality and only be used for the purpose of the research.
2. I understand the risks associated with taking part in this study.

3. The research project aim has been explained to me.
4. I do not have to respond to any question that I do not wish to answer for any reason.
5. Access to the records that pertain to my participation in the study will be restricted to persons directly involved in the research.
6. Any questions that I may have regarding the research, or related matters, or related matters, will be answered by the researcher.
7. Participation in this study is entirely voluntary and I can withdraw my participation at any stage.
8. I understood the information regarding my participation in the study and I agree to participate.

Signature of interviewee **Signature of witness**

---------- ----------

Signature of interviewer

APPENDIX C (ELLISRAS COMMUNITY LETTER)

UNIVERSITY OF LIMPOPO

Department of Physiology and Environmental Health

Private Bag x 1106
Sovenga
0727
SOUTH AFRICA
Tel: (015) 268 2953
Email: Kotsedi.monyeki@ul.ac.za
Website:www.ul.ac.za

29 October 2016

The Principal/Manager

……

……

……

Dear Sir/Madam

ALL ELLISRAS LONGITUDINAL STUDY MEMBERS ARE KINDLY REQUESTED TO TAKE PART DURING THE PERIOD NOVEMBER 2016 TO 5 JANUARY 2017

Diabetes, high blood pressure and cholesterol problems or high fat levels in the blood seem to be more common in the community than they were 10 or 20 years ago. This is most likely a result of both environmental and genetic factors. Environmental factors are those factors related to your lifestyle such as your diet and your physical activity levels. Genetic factors are those factors you "inherit" from your parents and grandparents. These conditions can lead to further health problems such as problems with eyesight, the heart and strokes, but the chance of having these problems can be lessened by treatment. It is my pleasure to report that the department of Physiology and Environmental Health, University of Limpopo will commence with the Ellisras Longitudinal Study (ELS) shortly. The aim of the ELS will be to track the role of lifestyle risk factors in determining adverse health outcomes. In particular, the development of non-communicable diseases, including obesity, hypertension, diabetes and coronary heart disease in a cohort of rural adolescents of South Africa over time.

All Ellisras Longitudinal Study subjects will be requested to take part. This research is looking at the adolescents' and young adults' lifestyle risk factors and how they may affect the development of non communicable diseases, including obesity, hypertension, diabetes, coronary heart disease. If you agree to participate, you will be asked about what and how much you had eaten the previous day. Anthropometric measurements will include weight, height, sitting height, leg length, skinfolds: (triceps, biceps, subscapular and supraspinale), girth (waist, hip, arm girth flexed and tense, neck, arm girth relaxed and calf girth), width: (femure and humerus). Your blood pressure and pulse rate will be measured.

Furthermore, after an overnight 10 hour fast (where you do not eat or drink anything except water after your evening meal, the night before) we will place a sterile little tube in a vein in your arm and take 15 mls (3 teaspoons) of blood. Taking the blood sample may cause a little discomfort at the site but there are no risks for this test, other than those associated with routine blood sampling. All procedures will be supervised and carried out by appropriately trained medical personnel from University of Limpopo, Medical Research Council, Vrije University Amsterdam, The Netherland le Sefako Makgatho Health Science University, who will use techniques to minimise any risks of infection. This test is used routinely for medical purposes. The blood sample will be used to determine your blood sugar, insulin, cholesterol and other additional factors that may help us learn more about diabetes and cardiovascular diseases risk factors.

These measurements and interviews will take place during the period November 2016 to 5 January 2017.

Please allow me to refer to PhD thesis of Monyeki (2000):

"…Bahlalerwa (cultural name of our population) I requested your help and you responded positively. I am happy because together we could make a difference. I am appealing to all of us to support and avail ourselves to any form of research activities taking place in our area. Such activities are geared towards improving the health not only of the Bahlalerwa population but the whole of South Africa if not Africa. We should keep focus and if somehow we could be blinded, with the help of the Almighty God the father of Jesus Christ, crystals like glass will fall from our eyes like what happened to Paul in the Holy Bible. Our vision will be broaden and we will no longer think and perform our duties inside the box…."

Pass my regards to everybody at home.

Yours sincerely

Prof Kotsedi Daniel Monyeki
Principal Investigator: Ellisras Longitudinal Study

Dr Marlise van Staden
Head of Department

APPENDIX D (PHYSICAL ACTIVITY QUESTIONNAIRE)
Ellisras Longitudinal Study (ELS)

2017- NORTHERN SOTHO VERSION
DEMOGRAPHIC, DIET AND PHYSICAL ACTIVITY DATA

1. Subject Number:
2. Code of the Fieldworker:
3. Examination Date:
4. Name of School/Village/Academic Institution :
5. School number/Village/Academic Institution number:
6. Last Grade/Level or Current Grade/Level at school :

7. Qualification/Profession	
8. Residential Address (Mo o dulang):	
9. Postal Address:	
10. Cell number:	Cell of close relative:

KAROLO YA DIJO KAROLO A				
1. Meetse ka moka ao a bedišitšweng a phepa go sa kgathale gore o a tšere kae?		nnete =1	maaka =2	
2. Seneke goba dijo tša magareng tše di loketšeng mmele ke				
	Galase ya maswi a tlhakantšhitšweng le dilwanalwana		1	
	Lefela leo le gadikilweng ka potoro		2	
	Tšhokolete		3	
	Karabo 2 le 3 mo godimo		4	
3. O ka nwa seno-tšididi bjale ka “coca cola” boemong bja meetse?		nnete =1	maaka =2	
4. Ga go kgonege gore o bone divitamine le dimenerale tšeo o di tlhokang mo dijong ka fao o swanetše go nwa dipilisi tša tšona.		nnete =1	maaka =2	
5. Sešebo sa soya se loketše mmele bjale ka nama		nnete =1	maaka =2	
6. Go itšhidolla go swanetše go ba karolo ya bophelo bja gago letšatši le lengwe le lengwe.		nnete =1	maaka =2	
7. Ge o e ja dijo tše dinago le phepo ga go hlokege gore o itšhidolle		nnete =1	maaka =2	
8. Swikiri goba dijo tše di nang le swikiri di kotsi mo mmeleng ka bjalo di swanetše go tlogelwa e le ruri.		nnete =1	maaka =2	
9. Dienywa le merogo di bohlokwa mo mmeleng wa gago		nnete =1	maaka =2	
10. Ke dienywa tše kae tše o swanetšego go di ja ka letšatši?				
	Dienywa tše 1 go fihla go tše 4		1	
	Dienywa tša go feta 5		2	
	Ga go hlokege go ja dienywa letšatši le lengwe le le lengwe		3	

11. Mehuta e mentši ya dijo e go fa di aga mmele go feta ge o e ja mohuta o tee.	nnete =1	maaka =2	
12. O swanetše ke go ja swikiri e ntšhi gore o be le maatla	nnete =1	maaka =2	
13. Mmele wa gago o hloka letswai le le nnyane gore o itekanele	nnete =1	maaka =2	
14. Mehuta yohle ya dinawa tše di omišitšweng di swanetše go jewa kgafetša kgafetša.	nnete =1	maaka =2	
15. Meetse a go tšwa mo meeding ya tlhago a bolokegile go nwewa	nnete =1	maaka =2	
16. Swikiri le dijo tšeo di na go le swikiri di swanetše go jewa gannyane	nnete =1	maaka =2	
17. Ke mokgwa o fe wa go apea wo o ka dirago gore dijo tša gago di be le makhura a mantši?			
	Go bediša	1	
	Go beša	2	
	Go gadika	3	
18. Dijo tša go swana le borotho, reisi, bogobe goba setampa di swanetše go ba karolo e kgolo ya dijo tšeo o di jang	nnete=1	maaka =2	
19. Letswai le swanetše go nokwa dijong tšohle ka ntle le dienywa fela	nnete =1	maaka =2	
20. Ke tsela e fe ya go apea ye o ka feleletšang o na le makhura a mannyane mo maeng?			
	Go apea ka meetse	1	
	Go gadika	2	
	Go bediša	3	
	Ga go tsela ya nnete go tšeo di lego mo godimo	4	
21. O ka nna wa ja nama e ntši ka moo o ratago letšatši le lengwe le le lengwe ka ntle ga go ba le ditlamorago tše di mpe.	nnete =1	maaka =2	
22. Ke dijo tše dife gare ga tšeo di latelang tšeo di nago le makhura a mannyane?	Disimba	1	
	Mmidi o o gadikilweng	2	
	Dichips	3	
23. Go bolokegile go ja dijo tša magareng tšeo di nago le swikiri e ntši	nnete=1	maaka =2	
24. Dijo tšeo di loketšego mmele….			
Ka mehla di na le nama e ntši, le setatšhe, dienywa, merogo le ditšweletšwa tša maswi tše di nnyane.		1	
Di na le merogo kudu le ditšweletšwa tša maswi tše di nnyane le nama ye nnyane.		2	
Ka mehla di na le setatšhe, dienywa le merogo kudu, le ditšweletšwa tša maswi tše di nnyane.		3	
Ga go na ya tšeo di leng ka godimo		4	
25. Go a thuša go ja dijo tše di nago le swkiri fela e le diseneke go na le gore e be karolo ya dijo.	nnete =1	maaka =2	
26. Dijo tše di nago le swikiri e ntši di swanetše go jewa bjale ka dijo tša magareng.	nnete =1	maaka =2	
27. O nwa meetse a makakang ka letšatši?			
Ga go hlokege go nwa meetse letšatši le lengwe le le lengwe		1	
galase e tee go ya go tše tharo		2	
digalase tše nne go ya go tše tshela		3	
digalase tše šupa go ya go tše seswai		4	
28. Ke maswi a makakang a o swanetšego go a nwa ka letšatši?			

	Ga go hlokege	1	
	bogare bja komiki	2	
	komiki e tee	3	
	dikomiki tše pedi	4	
29. Tsela ye e itekanetšego ya go ja ke			
	go ja dijo tše di farologaneng	1	
	go ja dijo tše dingwe gantši go fetiša tše dingwe	2	
	go ja mehuta ya dijo tše dingwe ka go lekana	3	
	tšohle tša ka godimo.	4	
30. Ke sehlopha sefe sa dijo seo o ka se jago ka bontši letšatši le lengwe le le lengwe?			
	borotho, setampa, reisi, bogobe	1	
	diapola, dipanana, sepinatšhe le dikherotse	2	
	maswi, yokate, tšheese	3	
	nama ya kgogo,hlapi, dinawa le mae	4	
31. Dijo tše di itekanatšego ke tšeo di nago le...			
	bontši bja nama, hlapi le kgogo	1	
	dijo tšeo gantši di sa apeiwago	2	
	dijo tšeo di leng mo ditšhitswaneng	3	
	gantši borotho, reisi le bogobe	4	
32. Swikiri e e itekanetšego ga bjalo e swanetše go hlakanywa le dijo tšohle	Nnete=1	Maaka=2	
33. Ke tše dife tša dijo tša mo mesong tšeo di nago le makhura a mannyane			
	Borotho bja korong bo bo bešitšwego le majerini	1	
	Weet-bix le maswi ao a nago le makhura a diperesente tše pedi (2%)	2	
	Nama ya kolobe le mae	3	
	Kgetho ya ka ke 1 le 2 mo godimo	4	
34. Dijo tše dintši tšeo o di jago di swanetše go ba le…			
	setatšhe, ditšweletšwa tša maswi, nama le dinawa	1	
	merogo, dienywa le ditšweletšwa tša maswi	2	
	setatšhe	3	
	merogo, dienywa, nama le dinawa	4	
35. Swikiri bontši e na le dikotla/divitamini le dimenerala	Nnete=1	Maaka=2	
36. Makhura goba dijo tša makhura di ka jewa ka tekano e nnyane	Nnete=1	Maaka=2	
37. Mehuta yohle ya dinawa e na le phepo kudu go ka jewa mo maemong a nama	Nnete=1	Maaka=2	
38. O ka nna wa nwa beini, bjalwa, disaeta tše dintši ka mo o ka kgonang ka gona, ga fela o jele pele	Nnete=1	Maaka=2	
39. Go nwa meetse a mantši ga go bohlokwa	Nnete=1	Maaka=2	

KAROLO YA B
GO ITSHIDILLA MMELE

Ipusantshetse ya ditirišo ya tše di latelago tšeo o di dirilego. Fa o apeile ka labobedi tshwaya 1 gaufi le mošomo, fa o apeile ka mosupologo, laboraro le labotlhano tshwaya 3 gaufi le mošomo.

A. Gare ga beke (Mosupologo o fihla Labotlhano) ke dira tse di latelago tša lelapa		0-5 score	
	Go apea		
	hlekisa ntlo		
	hlatswa dibjana di bona		
	phapha dikgong		

	hlokomela diruiwa		
	Dira temo		
	Rwala meetse		
B. Gare ga beke (Mosupulogo - Labotlhano) ke raloka meraloko e e latelago ya setso ga kae		0-5 score	
	Masekitlana		
	Morabaraba		
	Luto		
	Kgati/		
	Mmino		
	Sekonopa		
	Maphitlaphitlane		
	Diketo		
	Sekonopa		
	Molentse/Ulo		
	Sekapukapu		
	Mokoko		
	Mmela		
	Sepini		
	Pekwa		
	Katsekatse Legotlo		
	Legotlo		
	Setimela		
	Sekotiwa		
	Mambalobalo		
	Banaka		
	Mengwe tlhalosa		
C. Ke itshidilla ga kae ka meraloko ye mo bekeng (Mosupologo go fihla Labotlhano)		0-5 score	
	Volli bolo		
	Drum majorettes		
	Thuto ya go itshidila		
	Kgwele ya maoto		
	Kgwele ya diatla		
	Mabelo		
	Karate		
	Mabole		
	Mengwe tlhalosa		
Ipusantshetse ya mošomo tša mmele mafelelong a beke – Mokibelo le Sontaga: Tlhagisa gore o dirile mešomo ye mo kae. Ge o apeile ga tee mo mafelelong a beke ngwala 1 gaufi le mošomo. Ge o apeile ka Sontaga le ka Mashppologo ngwala 2 gaufi le mošomo eo.			
D. Mafelelong a beke (Lamatlhatso – Lamorena) ke dira mešomo ye e latelago ga kae		0-2 score	
	Apeya		
	Phepaphapha ntlo		
	Tlhatswa dijana		
	Tlhatswa diaparo		
	Phapha dikgong		
	Tlhokomelo ya leruo		
	Temo/bolemi		
	Thotha meetse		
E. Mafelelong a beke (Lamatlhatso le lamorena) ke itshidilla meraloko ye ga kae			
	Voli bolo		
	Drum majoretts		
	Thuto ya ikatiso		
	kgwele ya tshelela		
	Kgwele ya diatla		

	Mabelo			
	Karate			
	Mabole			
	Mengwe (tlhalolosa)			
F.	Bekeng ye e fedileng (matšatši a le 7) o ile wa ikatiša goba wa tseya karolo mo go itšhidolleng mmele bonnye metsotso e le 20 bjalo ka kgwele ya maoto, crikhethe, rugby, kgwele ya diatla, tlhokomelo ya diruiwa goba go siana			
	Ga se ke tseye karolo		1	
	Letšatši le le tee		2	
	Matšatši a le mabedi		3	
	Matšatši a le mararo		4	
	Matšatši a le mane		5	
	Matšatši a le mathlano		6	
	Matšatši a le tshelela		7	
	Letšatši ka letšatši		8	
G.	Bekeng ya go feta (matšatši a le 7) lebaka le legolo ke lefe la go go kgoreletsa go tseya karolo mo go itshidollong mmele			
	Ke tsere karolo mo itshidilong mmele mo bekeng (matšatši a le 7 a a fedileng)		1	
	Ke ne ke lwala		2	
	Ke ikutlwile ke sa bolokega, ke tshogile go ya lepatlelong, boitshidillong go tseya karolo.		3	
	Ga rena ditlabakelo, boikatisetso, lepatlelo moo re ka tseyago karolo ya go itshidolla mmele		4	
H.	Mo bekeng ye e fetileng (matšatši a le 7) o tsere karolo ga kae go ka go itshidilla mmele bonnye metsotso e le 30 go tse bjalo ka (Go pagama paesekela ka biketlo, go kgarametša mochene wa go sega bjang, go phimoloa mabota, go tšhasa goba go fiela mabota).			
	Ga se ke tseye karolo mo ikatisong ya mmele		1	
	Letšatši lele 1		2	
	Matšatši a le 2		3	
	Matšatši a le 3		4	
	Matšatši a le 4		5	
	Matšatši a le 5		6	
	Matšatši a le 7		7	
I.	GO ITSHIDILLA MMELE: Dipotšišo tše di latelang di mabapi le nako yeo o e tseyang go itshidilla mmele. Tšeo di akaretša mešomo yeo o dirago ka gae, mošomong, go tloga lefelong lengwe go ya go le lengwe le nako yeo e beetsweng go dira seo.O kgopelwa go araba dipotšišo le ge o itseya o se motho yo o mafolofolo			
	Maemo a kamanyo le mošomo (di lefelwa goba di sa lefelwe)			
1.	A mošomo wa gago o akaretša gantši gonna, go ema, goba go sepela nako e kopana (tlase ga metsotso e 10)	Eeng=1	Aowa=2	ga go kgonege=3
2.	A mošomo wa gago o akaretša mediro e boima bjalo ka (go kuka boima, go epa, goba e boima) bonnye metsotso e le lesome (10 minutes)	Eeng=1	Aowa=2	ga go kgonege=3
3.	Mo bekeng ya tlwaelo ke matšatši a makae ao mo go ona o šomago mešomo ye boima bjalo ka karolo ya mošomo wa gago?			Palo ya matšatši= ……
4.	Mo letšatšing la tlwaelo ke letšatši lefe mo o šomago mešomo ye boima. O tseya nako e kaakang go dira mošomo wo bjalo?			_____ diure ____metsotso
5.	A mošomo wa gago o akaretsa go dira ka maatla a a lekaneng bjalo ka go rwala merwalo e e bofefo ganyenyane metsotso e lesome	Eeng=1	Aowa=2	ga go kgonege=3
6.	Mo bekeng ya tlwaelo, ke matšatši a makae ao mo go ona o šomago mešomo ya maatla a a lekaneng bjalo ka karolo ya mošomo wa gago?			Palo ya matšatši = ……
7.	Mo letšatšing la tlwaelo ke lefe leo o dirileng mošomo wa maatla a go lekanela, o tseya nako e kakaang go dira mošomo e bjalo			_____ diura ____metsotso

8.	Letšatši la gago la mošomo ke botelele bjo bo kaakang?			_____ diura ____metsotso	
	Mošomo wo o amago go itshidilla ga mmele. Kwa ntle ga mošomo wo o setsego o o tlhagisitse, ke rata go go botšiša ka ga mokgwa go tloga lefelong le lengwe go ya go le lengwe (mošomong, kerekeng, lebenkeleng, mmaketeng)				
9.	A ge o tsamaya, o šomiša peretshitswana bonnyane metsotso e lesome go ya le go boa lefelong?	Eeng=1	Aowa=2	ga go kgonege=3	
10.	Mo bekeng ya tlwaelo, ke mo matšatšing a makae ao mo go ona o tsamayang ka peretshitšawana bo nnyane metsotso e lesome go ya le go boa lefelong?			Palo ya metsotso= ……	
11.	Letšatšing la tlwaelo, o tseya nako e kaakang go sepela goba go namela peretshitšawana ge o le mo tseleng?			_____ diura ____metsotso	
	Go itshidilla mmele go tšamaelang le nako ya maikhutso. *Dipotšišo tse di latelang di ka go itshidilla mo nakong ya maikhutšo goba e e beetsweng thoko (lebelela karata). O seke wa akaretša go itšidilla ge o le mo mošomong goba go ya mošomong ka ge re setse re boletše ka gona*				
12.	Mo nakong ya gago ya maikhutšo goba e e beetsweng thoko, o tle o itshidilla boima goba ka maatla a a lekaneng moo go go o tseyago metsotso e le lesome ka nako?	Eeng=1	Aowa=2	ga go kgonege=3	
13.	Mo nakong ya gago ya maikhutšo goba e e beetsweng thoko, a o tle o dire mošomo wa boima (bjalo ka go kitima, meraloko ye e boima goba go kuka dithšipi) bonnyane metsotso e le lesome ka nako?	Eeng=1	Aowa=2	ga go kgonege=3	
14.	Mo bekeng ya tlwaelo, ke matšatši a makae mo o šomago mešomo ye boima bjalo ka karolo nako ya maikhutšo goba ye e beetsweng thoko?			Palo ya matšatši = ……	
15.	O šomiša nako e kaakang go šoma mešomo ye boima mo letšatšing la tlwaelo?			_____ diura ____metsotso	
16.	Mo nakong ya gago ya maikhutšo goba e e beetsweng ka thoko a o dira mošomo wo maatla goba wo o lekanego bjalo ka go sepela ka mafolofolo, go rutha gannyane metsotso e le lesome?	Eeng=1	Aowa=2	ga go kgonege=3	
17.	O tšea matšatši a makae go dira mošomo wo maatla goba wo o lekaneng bjalo ka karolo ya nako ya maikhutšo goba ye e beetšego ka thoko.			Palo ya matšatši = ……	
18.	O tšea nako ye kaakang go dira mošomo wa maatla a a lekaneng, mo letšatšing la tlwaelo?			_____ diura ____metsotso	
	Bjale ke rata go go botšiša ka nako ye o e šomišago go dula le go khutša go sa akaretse go robala, matšatši a le šupa a a fetilego. Se, se ka akaretša nako e e dirisitsweng go nna tafoleng, go etela bagwera, go bala goba go dula fase o lebeletse TV.				
19.	Mo matšatšing a le šupa a a fetileng o tsere nako e kae o ntse fa fase goba o patlame, mo letšatšing la tlwaelo?			_____ diura ____metsotso	
	Mešomo ya ditlhopha tša meraloko				
1.	A o ikamaganya le mekgatlho ya meraloko ge o ikatišago, bjalo ka kgwele ya dinao goba ya diatla?	Eeng=1	Aowa=2		
2.	Ke ka makgehlo a makae mo o ikatišago le sehlopha				
	hlalosa papadi	Diri…….. ka nako e le tee			
	hlalosa papadi	Diri……ka nako e tee			
	hlalosa papadi	Diri……ka nako e tee			
	hlalosa papadi	Diri……ka nako e tee			

3. Nako ya go ikatiša le sehlopha			
	Letšatši le letšatši	1	
	Gararo- gane mo bekeng	2	
	Ga bedi mo bekeng	3	
	Ga tee mo bekeng	4	
	Ga tee goya gabedi mo kgweding	5	
	Ga ke tsebe	6	
4. Ge o sepela, o sepela ka lebelo le le kaakang ka tlwaelo?			
	Ka lebelo le le boima la go dirago gore ke heme boima go feta ka tlwaelo	1	
	Ka lebelo la mahareng le le dirago gore ke heme boima go feta ka tlwaelo	2	
	Ka lebelo la tlase mo go senago phetogo	3	
5. Ge o raloka o hema ka boima bjo bo kaakang?			
	Ka lebelo le le boima la go dirago gore ke heme boima go feta ka tlwaelo	1	
	Ka lebelo la mahareng le le dirago gore ke heme boima go feta ka tlwaelo	2	
	Ka lebelo la tlase mo go senago phetogo	3	

KA MO MALWETŠE LE GO TLHOKOFALA, GO TLHAGELELANG KA GONA MO LEPHALALE

A o na le maloko a gaufi? (Batsofadi, batswadi, dikgaetsedi le bo morwa rre) ao nako enngwe ba ileng ba nna le malwetše ao a latelago

	swikiri		kgatelo e godimo ya madi		Go tlhaselwa ke pelo		kholesterolo		seterouku		
	Eeng=1	Aowa=2	Eeng=1	Aowa=2	Eeng=1	Aowa=2	Eeng=1	Aowa=2	Eeng=1	Aowa=2	
Batsofadi											
Batswadi											
Dikgaetsedi											
Malome											
Mmane											

KAROLOR YA C
MAEMO A DEMOKRAFIKI LE SOCIO-EKONOMI

O HUDUGILE GA KAHE LE GONA O NE O NNA KAE?

				OFFICE USE ONLY
1. Naa, o dutse lebaka le le kaakang mo atereseng ye o dulago gona bjale? Ngwala mengwaga goba dikgwedi		Me/ngwaga	Di/kgwedi	
2. Naa, o be o dula kae pele?				
	Lekeišeneng le lengwe Lephalale	1		
	Motse seteropo Lephalale	2		
	Motse selegae o ke nnang go ona bjale	3		
	Mo motseng mongwe mo Lephalale	4		
	Mo motseng mongwe ka ntle ga Lephalale	5		
3. Ke nako e kang kang o dula mo bodulong bja gago ba bjale		Dingwaga	Dikgwedi	
4. O belegetsw kae?		Dingwaga	Dikgwedi	
MAEMO A MOŠOMO				
1. A o a šoma		Eeng=1	Aowa=2	
2. E fa leina la mong mošomo, kgwebo, goba kontraka				
	Medupi	1		
	Matimba	2		
	Iscor/Iskoro	3		
	mo marekelong	4		
	Dingwe/thlalosa……	5		
3. Na o šoma mošomo wa mohuta mang? Hlalosa ka boripana:		Ga ke šome		
4. E fa maemo a gago mošomong ……		Ga ke šome		
5. Naa o ile wa šutišiwa mošomong wa gago dikgweding tše tshelelago tše di fitilego?	Eeng=1	Aowa=2	Ga ke šome	
5.1 Ge karabo e le Eeng, ke ka lebaka lefe? ……				
6. Hlalosa mošomo wa gago wa pele gayooo šomang gona bjale		Ga ke šome		
7. Efa leina la setlamo goba la kgwebo:……		Ga ke šome		
8. O šomiša eng go ya mošong?		Ga ke šome		
	Bese	1		
	Tekisi	2		
	Koloi ya poraifete	3		
	O sepela ka Maoto	4		
	Kariki ya ditonki	5		
	Ka tše dingwe hlalosa……..	6		
9. O ya mošomong ka nako mang?	Ga ke šome	1		
	3-4:59 phakela	2		
	5-6:59 phakela	3		
	7-9 phakela	4		
	Tse dingwe	5	……..	
10. O thoma go šoma ka nako mang?	Ga ke šome	1		
	5-6:59 phakela	2		
	7-8:59 phakela	3		
	Tse dingwe	4	……..	
11. A o fihla gae ka nako mang ge o tšwa mošong?	Ga ke šome	1		

	15-16:59 thapama	2		
	17-18:59 thapama	3		
	Tse dingwe	4	……	

12. Go na le mokgatlo wa bašomi mošomong wa gago?	Eeng=1	Aowa=2	Ga ke šome	
13. A le šoma ditšhifi sekofo?	Eeng=1	Aowa=2	Ga ke šome	

14. Ke ditšhifi dife kwa mošomong?	Diiri tse 8 =1	Diiri tše 12=2	Tše dingwe =3	Ga ke šome	
15. Wena o tsena sekofo se efeng?	Motshegare=1	Bošigo =2	Tatelano = 3	Ga ke šome	
16. A o lefiwa ka kgwedi goba ka beke mo o šomago?		Kgwedi =1	Beke =2	Ga ke šome	

17. Palo ya bao o dulago nabo

		Banna/basimane	Basadi/basetšane
	Bana ka fase ga ngwaga		
	Bana ba mengwago ya 1 – 6		
	Bana ba mengwago ya 7-12		
	Bana ba mengwago ya 13-18		
	Babagolo ba dingwaga tše 18-30		
	Babagolo ba dingwaga tše 31-45		
	Babagolo ba dingwaga tše 46-60		
	Babagolo ba dingwaga tše 61 le go feta		

TLINIKI YA KGAUFI

Leina la tliniki	Mohuta wa dinamelwa go ya tliniking	O tšea nako e kakang go fitlha tliniking …….....	
	oSepela ka maoto = 1	Metsotso/di iri tse kae	
	Tekisi = 2	Metsotso/di iri tse kae	
	Koloi ya gago/mogae = 3	Metsotso/di iri tse kae	
	Tse Dingwe tlhalosa ……		

THUTO

1. Polelo ya geno ke efe?	Sotho	1
	Tswana	2
	Tsonga	3
	Xhosa	4
	Zulu	5
	Venda	6
	Others (specify)	7……

2. Thuto ya godimo ye o tšweletšego mo go yona		
	Ga se ke tsene sekolo	
	mphato 1-2	1
	mphato 3-5	2
	mphato 6-7	3
	Mphato 8	4
	mphato 9	5
	mphato10	6
	mphato11	7
	mphato12	8

3.1 O na le mangwalo a ka godimo ga mphato wa lesomepedi	Eeng=1	Aowa=2
3.2 O tsena sekolo kae gona bjale? ……	Ga ke tsene sekolo=1	

3.2 O bala mphato o feng gona bjale? ………………………………………			Ga ke tsene sekolo = 1		
4. E fa lebaka la go dira gore o tlogele sekolo……………………………………………………………………………					
5. Bontšha mangwalo ao o nago le ona a dithuto tša godimo					
	Ga kena yona	1			
	Ga ke a fetša diploma	2			
	Diploma	3			
	Diploma ya kwa kholetšheng	4			
	Diploma ya thekinikono	5			
	Dikiri ya univesiti	6			
	Tse Dingwe tlhalos……………………………………	7			

DIPOTŠIŠO KAKARETŠO KA TŠA KA LAPENG

1. Le humana meetse a go nwa ka gae go tšwa kae				
	Peipe ya meetse le lapeng	1		
	Meetse ao a borileng	2		
	thepe ya bolhe	3		
	Koloi ya go rwala meetse	4		
	Letamo/ noka/ meedi/didiba	5		
	Meetse a pula	6		
	Tse Dingwe tlhalosa…………………	7		
2. Na go ka tšea nako e kaakang go fihla mo sedibeng sa meetse le go boela gae?				
3. Le šomiša ntlwana ya botshwelomare ya mohuta ofe				
	Ntlwana boithusetso ya meetse	1		
	Ntlwana boithusetso ya meetse ya go hlakanelwa	2		
	Ntlwana boithusetso ya mošima	3		
	Ga go na ntlwana boithusetso	4		
	Tše dingwe tlhalosša……………………………………	5		
4. Le šomišang eng ka lapeng go apea le go ruthetšameetse				
	Motlakase	1		
	Gase	2		
	Leokwane / parafeni	3		
	Dikgong	4		
	Malahla	5		
	Dišhu	6		
	Tše dingwe tlhalosa …………..	7		
5. Ngwako wa lena o na le di phapoši tše kae?		…………….diphapuši		
6. Ke diphapoši tše kae tša go robala?		…………….diphapuši		
7. Na wena goba o mongwe ka mo gae o hwetša dijo/tšhelete tša mphiwa fela		Eeng=1	Aowa=2	
7.1 Ke ba ba kae ka mo gae bao ba hwetšago dijo/tšhelete tša mphiwa fela		…………… batho		
8. A o na le tšhingwana ya merogo? Eeng=1 / Aowa=2 Ge karabo e le Aowa, e fa lebaka,,				

9. Ke ka makgetho a le makae moo ba lelapa la gago/geno ba robalang ka tlala?	Gantši = 1	dinako tse dingwe = 2	Ga se gantši = 3	Ga nke ba robala ka tlala = 4	
10. Ke ka makgetho a le makae moo ba lelapa la gago/geno ba swanelang ke go	Gantši = 1	dinako tse dingwe = 2	Ga se gantši = 3	Ga nke ba robala ka tlala = 4	

fokotsa kelo ya dijo bakeng la dijo tša go se lekane					
11. A lelapa la gago/geno le na le tše o di latelang?					
	Motlakase	1	Eeng=1	Aowa=2	
	Seyalemoya	2	Eeng=1	Aowa=2	
	setlhagisa ditshwantsho (TV)	3	Eeng=1	Aowa=2	
	Telephone (land-phone)	4	Eeng=1	Aowa=2	
	Sellathekeng (cell)	5	Eeng=1	Aowa=2	
	Setšidifatši	6	Eeng=1	Aowa=2	
	mochene wa go tlhatswa	7	Eeng=1	Aowa=2	
	khomputa	8	Eeng=1	Aowa=2	
	setofo sa malahla	9	Eeng=1	Aowa=2	
	setofo sa sethuthufatsi	10	Eeng=1	Aowa=2	
	Se thuthufatsi	11	Eeng=1	Aowa=2	
	setofo sa parafene	12	Eeng=1	Aowa=2	
12.A e ka ba o mongwe ka mo gae o na le tše dingwe tša di diriswa tše di latelago:					
	Sefatanaga	1	Eeng=1	Aowa=2	
	sethuthuthu	2	Eeng=1	Aowa=2	
	peretshitswana/ paesekele	3	Eeng=1	Aowa=2	
	kariki ya ditonki/ Mmeila	4	Eeng=1	Aowa=2	
	kariki ya go goga ka diatla	5	Eeng=1	Aowa=2	
	Kiribane	6	Eeng=1	Aowa=2	
	tonki/ pere/ mmeila	7	Eeng=1	Aowa=2	
	Dinku/dikgomo/dipudi	8	Eeng=1	Aowa=2	
	dikolobe	9	Eeng=1	Aowa=2	
	Dikgogo	10	Eeng=1	Aowa=2	
	Diphofolo tse dingwe	11	Eeng=1	Aowa=2	
13. E kaba o mongwe wa leloko o rekiša tše dingwe tša dibjalo? Ge karabo ya gago e le Ee bolela tše dingwe tša dibjalo tšeo. ……			Eeng=1	Aowa=2	
14. A go mongwe wa lelapa yo a bjalang le go rekiša go bona tselete Ge karabo e le Eeng hlalasa mehuta ya tšeo di rekišwago. ……			Eeng=1	Aowa=2	
15. Mehuta ya ditshenyagalelo ka morajo ga kgwedi tše nne:					
	Ditefo tša sekolo	1	Ee, bokae……	Aowa=2	
	Ditefo tša kalafo	2	Ee, bokae……	Aowa=2	
	Ditefo tša kalafo ya leruo	3	Ee, bokae……	Aowa=2	
	temo	4	Ee, bokae……	Aowa=2	
	Ditefo tša ka gae	5	Ee, bokae……	Aowa=2	
	Ditefo tša bašomi	6	Ee, bokae……	Aowa=2	

	rente	7	Ee, bokae……..	Aowa=2	
	makgetho	8	Ee, bokae……..	Aowa=2	
	Moneelo	9	Ee, bokae……..	Aowa=2	
	Tše dingwe hlalosa……..	10	Ee, bokae……..	Aowa=2	
16. Le šomiša tšhelete e kaakang ge le reka dijo ka beke?				Bokae…..	
17. Ke batho ba bakae bao ba tlišago letseno ka gae?				Bokae…..	
18. Letseno la bona ke bokae ka kgwedi (rente, mogolo, dithekišo, mphiwafela, le tse dingwe, etc.)	Ga ke diri			1	
	Mogolo			2	
	Thekiso			3	
	Motente			4	
	Tse dingwe, hlalosa ……..			5	
19. A e ka ba letseno ke lona la ka mehla la ka gae naa?			Eeng=1	Aowa=2	
20. A e ka ba letseno le le ka godingwana goba ka fasana gala dikgweding tše tshela tše di fitileng?			Eeng=1	Aowa=2	
21. Ge re bapetša letseno le, le la ngwaga o fitileng a re bona bophelo bo le kaone ka mo gae?					
	ikutlwa bokaone?			1	
	ikutlwa go tshwana le mengwageng?			2	
	Ke kwa bokete le go feta?			3	

PEER REVIEWED ARTICLES EMANATING FROM THE DISSERTATION

1. Matshipi M, Monyeki KD, Kemper HCG. (2017). The Relationship between Physical Activity and Plasma Glucose Level amongst Ellisras Rural Young Adult Males and Females: Ellisras Longitudinal *International Journal of Environvironmental Research and Public Health* 14(2):198p.

2. Matshipi M, Monyeki KD, Kemper HCG, Choma SSR, Makgopa HM. Association of physical activity with insulin resistance and diabetes in young adults: The Ellisras Longitudinal Study. Submitted to *BMC Public Health*, 2018. (Received favourable peer review comments)